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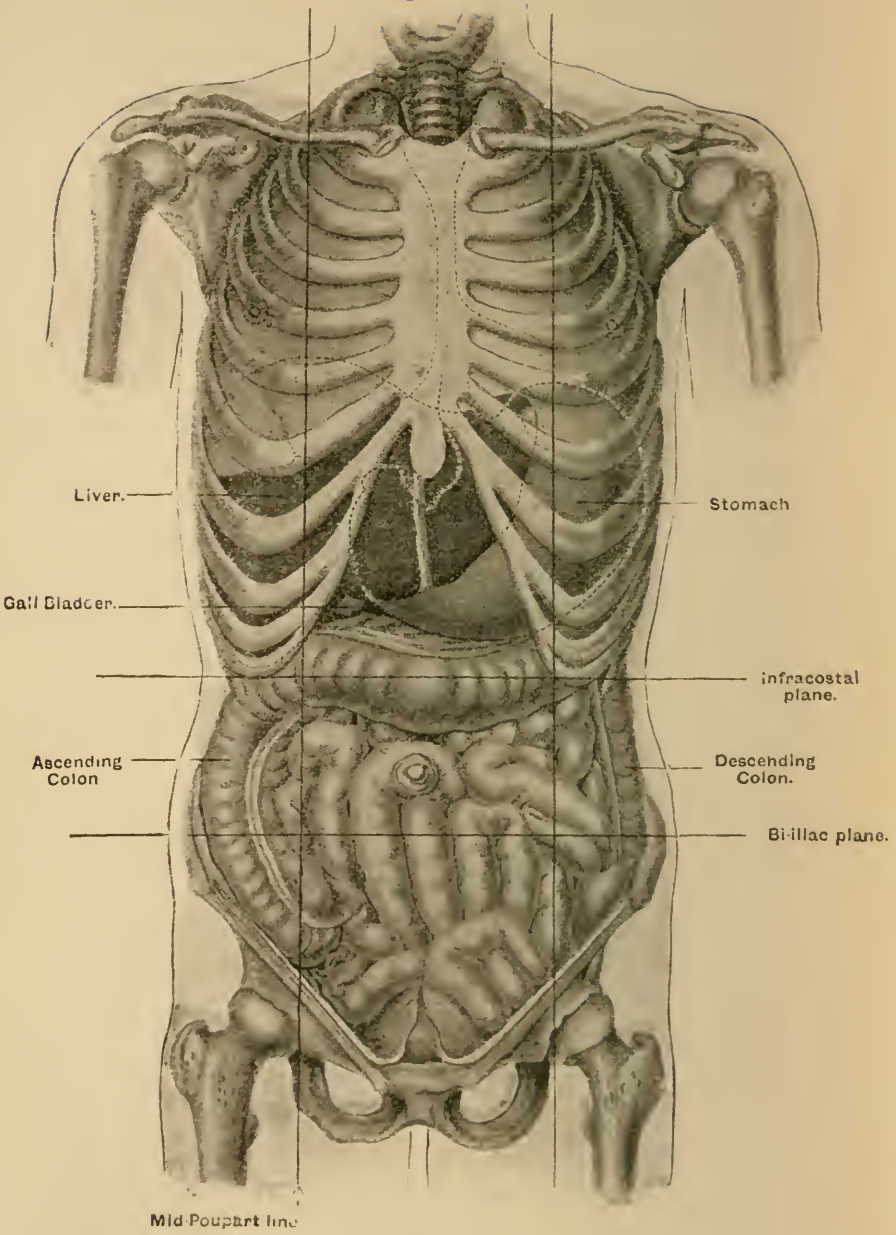

PLATE I.

FRONT VIEW OF ABDOMINAL VISCERA.
(After Luschka.)

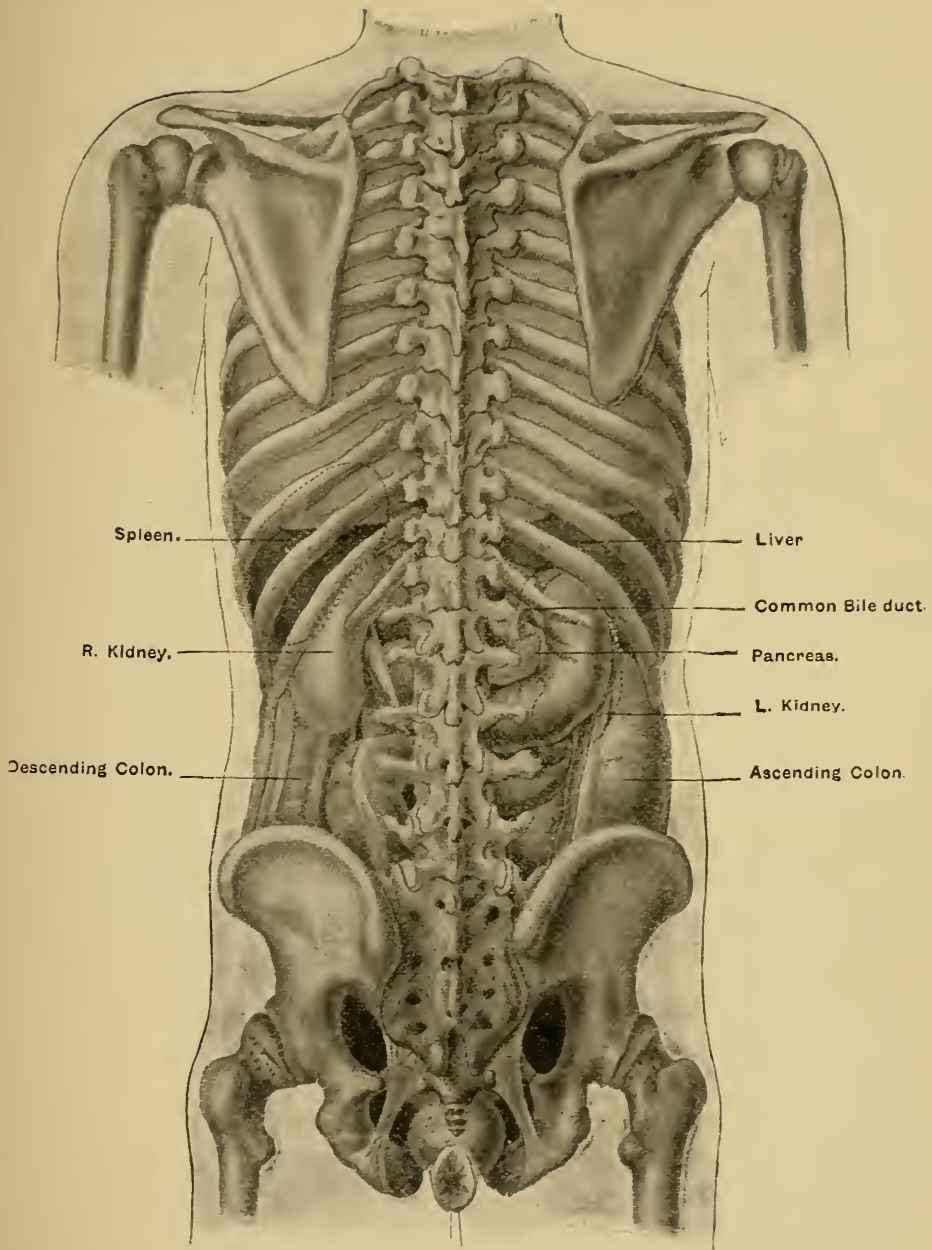

PLATE II.

BACK VIEW OF ABDOMINAL VISCERA.

(*After Luschka.*)

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A

Manual of Medicine

EDITED BY

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VOL. V.

DISEASES OF THE DIGESTIVE SYSTEM AND OF THE LIVER
DISEASES OF THE PERITONEUM AND OF THE VESSELS OF THE ABDOMEN
DISEASES OF THE KIDNEYS
DISEASES OF THE DUCTLESS GLANDS

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DISEASES OF THE DIGESTIVE ORGANS

THE GENERAL ANATOMY AND PHYSIOLOGY OF THE DIGESTIVE ORGANS

DIGESTION is that physico-chemical process whereby the food is converted into a fluid and diffusible condition suitable for absorption directly or indirectly (*viâ* the lacteals) into the blood-current. It is in some measure anticipated by, and is a continuance of, the changes which are effected by cooking. Some of the food taken is absorbable at once, whilst other constituents are incapable of solution in the alimentary canal, and are consequently indigestible. The digestibility of food, therefore, depends on the facility and completeness with which it is reduced to a diffusible state; its nutritive value, which is a different matter, being in proportion to its suitability for tissue-construction and metabolism.

For the carrying out of the function there are required various solvent juices to deal with the several kinds of alimentary principles, some arrangement by which these fluids and the food are mixed and propelled, and an adequate removal of the digested food-stuffs (digesta), an accumulation of which interferes with the proper progress of the digestive changes. These requirements are met (i.) by the alimentary canal, which is a tube of great length, presenting various alterations in size and situation in its course, which allow such opportunities for delay in the progress of the contents as are necessary for the efficient action of the solvents, and connected with which is an arrangement of contractile muscular tissue so disposed as to effect the complex movements of mastication, deglutition, gastro-intestinal peristalsis and defæcation; (ii.) by the secretions of the salivary, gastric, pancreatic, and intestinal glands, which are abundantly poured into the canal; (iii.) an extended absorbent surface, which is furnished by the mucous membrane with its vast area of valvulæ conniventes, villi, etc., where the blood capillaries and lacteal radicals are brought into close relation with the digested material; (iv.) a sufficient blood and lymph supply; and (v.) a con-

trolling and co-ordinating nervous mechanism, which is derived from the cerebrospinal and sympathetic systems, as well as an intrinsic arrangement of ganglia and fibres.

The Normal Anatomy of the Alimentary Canal

The passage through the trunk from mouth to anus which the solid and liquid ingesta traverse constitutes a canal upwards of 30 feet in length—five to six times the height of the individual. The relative length and capacity of the canal to the body height have some reference to the nature of the food, being much shorter in the carnivora and longer and more capacious in the strictly vegetable feeding animals. To permit of the necessary subdivision of the solid food, and of the efficient action of the digestive fluids, the canal offers special arrangements, such as the mouth, stomach, and dependent coils of the small intestine; and at several points the passage of the contents is exposed to some check—greater in the reverse direction—by more or less perfect valves and windings of the tube, of which the pyloric, ileo-colic and rectal valves, the opening of the gullet into the stomach, the duodeno-jejunal junction, the saccules and flexures of the colon, and the sigmoid are the most important.

From the clinical as from the anatomical point of view the **mouth** is held to mean the cavity which is bounded above by the hard and soft palate, below by the tongue and the musculo-membranous structure known as the floor of the mouth, and laterally by the cheeks. It has two orifices—the anterior, formed by the lips, and the posterior, leading into the pharynx. Within the buccal cavity are the alveolar processes of the jaws covered by the mucous lining and a dense connective tissue constituting the gums, in sockets of which are the teeth. Opening into the mouth are the ducts of the three pairs of salivary glands—parotid, submaxillary, and sublingual.

Between the mouth and the gullet is the oral part of the **pharynx**, which is a funnel-shaped tube lying close in front of the bodies of the upper cervical vertebræ and behind the mouth and larynx, and is about $3\frac{1}{2}$ inches in length. The communication with the mouth, known as the isthmus of the fauces, is formed on each side by two folds of mucous membrane covering muscles which pass downwards from the soft palate, the anterior to the tongue, and the posterior to blend with the lateral wall of the pharynx; between these folds or arches are the tonsils.

The **œsophagus** is the connecting tube between the pharynx and the stomach. It is $9\frac{1}{2}$ inches long, extending from the disc between the sixth and seventh cervical vertebræ to the upper border of the eleventh dorsal. Its upper end is on a level with the lower border of the cricoid cartilage and 6 inches from the incisor teeth; the cardiac

orifice of the stomach is hence $15\frac{1}{2}$ inches from these teeth. The average diameter of the tube is about an inch; it is narrowed in three places, viz. at its two extremities, and at a point $2\frac{3}{4}$ inches from its commencement, where it is crossed by the left primary bronchus. This point marks the site of the diverticulum given off by the primary fore-gut to form the lungs. The course of the œsophagus is not perfectly straight, but somewhat sinuous; commencing in the middle line, it deviates slightly to the left, returns to the middle at the level of the fifth dorsal vertebra, and inclines again to the left in the remainder of its length; besides these lateral bends it is also curved forwards, following the direction of the vertebræ. It lies behind the trachea, left bronchus, and left auricle, between the pleuræ and in front of the descending aorta, thoracic duct, and vertebral column. On each side is the recurrent laryngeal nerve lying in the angle between the gullet and the trachea, the common carotid artery, a part of the thyroid body, and the pneumogastric nerves. After passing through the diaphragm the tube bends sharply to the left and enters the stomach at an angle.

The inner surface is normally paler than that of the pharynx or stomach, and exhibits numerous longitudinal folds formed by the loosely attached mucosa, which are obliterated as the tube is distended.

The **stomach** is the somewhat pear-shaped expansion of the alimentary canal continuous with the gullet immediately after that tube has passed through the diaphragm. It occupies the upper part of the abdominal cavity on the left side, lying in great part behind the ribs, the larger end reaching as high as the fifth rib or even the fourth left intercostal space, where it is in contact with the diaphragm, which separates it from the heart, and the lower end (pylorus) where it becomes continuous with the duodenum, lying to a variable extent to the right of the middle line according to the degree of distension of the organ. The general direction of the viscus is therefore obliquely downwards and forwards from left to right, being about 10 to 12 inches in greatest length in the average adult male when moderately distended. The orifices by which it communicates with the œsophagus (cardiac) and with the duodenum (pyloric) are from 3 to 5 inches apart, and situated on the upper or smaller curvature, so that the left portion of the organ constituting the fundus extends farther to the left and higher than the cardiac opening, which lies to the left side of the body of the tenth or eleventh dorsal vertebra outside the aorta, the pyloric orifice being lower and at the level of the right side of the body of the first lumbar vertebra nearer to the anterior abdominal wall. Inasmuch as the œsophageal opening is the most fixed point of the stomach, and the pylorus is freely movable, the relative positions of these openings to each other, from before back, is subject to much variation with the state of fulness of the organ. Whilst, therefore, the cardiac orifice remains fixed in position, the pylorus, which is usually just behind the anterior edge of the liver, and lies close to the

right of the median plane, 1 to 2 inches below the tip of the ensiform cartilage, when the stomach is empty, may be found 2 or 3 inches to the right when the organ is distended, and may then be in contact with the abdominal wall immediately below the liver. With the varying distension of the stomach the anterior and posterior surfaces, which are in apposition in the empty state, shift in position, the former coming to be directed more upwards instead of forwards as the organ dilates, with corresponding alteration in the posterior surface, the viscus rotating comewhat on a transverse axis.

The relations of the stomach to adjacent structures are represented in the following diagram, which has been furnished to the writer by Prof. Thane :—

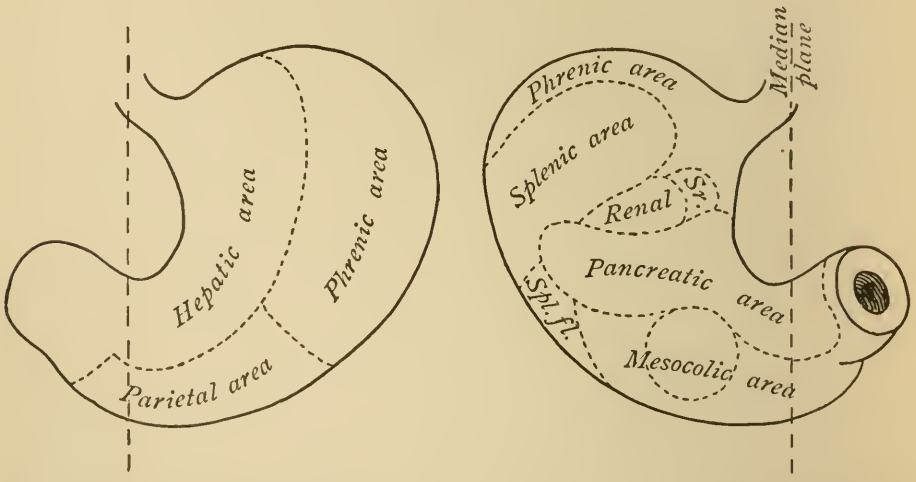

FIG. 1.—Showing the anterior and posterior relations of the stomach to adjacent structures. *Spl. fl.*, splenic flexure ; *Sr.*, suprarenal body. The circle on the right-hand figure indicates the position of the duodeno-jejunal flexure, which is separated from the stomach by the transverse meso-colon.

The most important surface relations, in addition to those already mentioned, are—the œsophageal opening nearly corresponds with the junction of the seventh left costal cartilage with the sternum from 4 to 5 inches from the surface ; the summit of the fundus is indicated on the surface by the lower border of the fifth rib in the mid-axillary line, or the upper border of the same rib in the nipple line ; the lowest normal limit of the greater curvature, which passes from under the liver close to the left of the gall-bladder, should not be below a line drawn $1\frac{1}{2}$ inches above the level of the umbilicus, and when the organ is empty the line may be considerably higher.

The normal capacity of the adult male stomach may be as much as 3 pints, but is usually less. The weight of the organ is about $4\frac{1}{2}$ ozs.

Some departures within normal limits from the above data are

met with, and the organ may be situated somewhat higher or lower; notwithstanding that it moves slightly with respiration, this is rarely recognised by examination.

Although the stomach ordinarily appears, when viewed externally, as a single pyriform bag, marked off at the pylorus by a circular constriction from the duodenum, it will be found that functionally regarded it consists of two parts, a cardiac and a pyloric, the latter being much the smaller and of about a fourth the capacity of the other. Occasionally indications of a separation by the appearance of a slight groove on one or both curvatures, several inches from the pylorus, and sometimes a thickening (of the muscular coat) may be felt along a line joining these grooves—pre-pyloric sphincter—and a deep constriction is now and then seen, due to the contraction of the muscular fibres in this situation, showing how the organ is imperfectly divided into two unequal portions, a condition which probably obtains to some degree during the process of gastric digestion.

As seen from the inside the surface of the living stomach is found to be of a deep rose-red colour, an appearance which is quite lost after death, being replaced by an ashy gray coloration not unfrequently marked by petechial spots. Unless much distended, the mucous membrane is thrown into numerous sinuous folds or rugæ, caused by a wrinkling of the loosely attached membrane as the muscular layers contract. These rugæ, which are mainly disposed in a longitudinal direction, are best marked along the greater curvature and less distinct towards the pylorus. The pyloric valve or sphincter is formed by a circular fold of mucous membrane covering a special thickening of the circular muscular layer, which projects into the lumen of the canal, leaving an aperture that admits the finger, and is capable of being more or less completely closed. A slight constriction externally marks the position of the pylorus, which may be felt as a distinct thickening.

The **duodenum** is the first portion of the intestine intervening between the stomach and the jejunum. It is about 10 to 12 inches in length and 2 to $2\frac{1}{2}$ inches in diameter, being in this latter respect somewhat larger than the rest of the small intestine. It forms a curve of a shape varying with the position of the pylorus, with the concavity, in which lies the head of the pancreas, directed to the left and somewhat upwards. It is conveniently described in four parts—the 1st being almost horizontal from left to right and before back, moving with the pylorus, and consequently being almost directly antero-posterior when the stomach is distended and the pylorus is moved to the right. Posteriorly are the common bile duct, the portal vein, and the gastro-duodenal artery, and in front it is overlapped by the liver. The 2nd portion, about 3 inches long, extends vertically and slightly backwards from the level of the right side of the body of the first lumbar vertebra, close under the neck of the gall-bladder, to the right side of the body of

the third or fourth lumbar, from which it is separated by the vena cava, with the right kidney to its outer side; in front are the under surface of the liver, and below that the transverse colon with which it is connected by the transverse meso-colon. The 3rd part, which forms an angular curve with the foregoing, and is 2 to 3 inches long, curves transversely and slightly upwards to the left side of the aorta, with which and the vena cava it is in contact, the inferior mesenteric vessels passing down in front between it and the transverse colon, and the head of the pancreas above. The last two inches of the duodenum, constituting the 4th part, bends upwards on the left side of the aorta to end by a sharp curve forwards in the jejunum, on the left side of the body of the second lumbar vertebra. Behind it is the psoas muscle and sometimes the inner edge of the left kidney; in front is the stomach, from which it is separated by the transverse meso-colon. This terminal part of the duodenum, however much the rest of this section of the canal may be displaced, maintains a uniformly fixed position owing to the presence of a musculo-fibrous band which attaches it to the left crus of the diaphragm, and this fixity is further aided by the post-peritoneal connective tissue.

Superficially the 2nd part is indicated by a line drawn vertically upwards from midway between the right anterior superior iliac spine and the middle line, in a line joining the two iliac spines (the right lateral line of Addison)[1]; and the angle of junction of the 2nd and 3rd parts mainly corresponds to the highest point of the iliac crest; and the duodeno-jejunal flexure lies opposite a point 3 to 4 inches above the umbilicus slightly to the left of the middle line.

The inner surface of the first couple of inches or so of the duodenum is smooth, but at that distance the valvulæ conniventes commence, becoming large and of regular concentric shape beyond the papilla which marks the common entrance of the hepatic and pancreatic ducts. The orifice of the combined ducts would admit a small probe.

The **jejunum** and **ileum**, which together measure about 22 feet in length and slightly diminish in calibre in their course, form numerous coils which lie in the middle and lower regions of the abdominal cavity and pelvis. This part of the canal is suspended to the spine by a reflection of the peritoneum—mesentery—which is attached for about 6 inches obliquely downwards "from the left side of the second lumbar vertebra, across the third part of the duodenum, aorta, inferior vena cava, and right psoas muscle to the right iliac fossa, where it ends" (Quain), spreading out to envelop the entire length of the small bowel. Between the layers of the mesentery pass the blood-vessels, lacteals, nerves, together with lymphatic glands and a variable amount of connective tissue and fat continuous with similar post-peritoneal

[1] *The Topographical Anatomy of the Abdominal Viscera*, by Prof. Addison, being the Hunterian lectures at the Royal College of Surgeons of England, 1901.

structures. The average length of the mesentery is about 8 inches, often becoming longer after middle life. As the last part of the ileum passes from the pelvic cavity upwards and outwards across the right psoas muscle to enter the colon it becomes more fixed, owing to a much shorter peritoneal attachment, which is often strengthened by a prominent peritoneal fold—plica vascularis—which passes downwards from beneath the ileum, as it overlies the inner border of the psoas, to the middle of Poupart's ligament. Sometimes this part of the bowel is firmly bound down to the iliac fossa, whilst in other cases it lies in the pelvic cavity with a prolapsed cæcum.

It would seem that the intestines, even after disturbance, do in a general way fall back again into their previous position, and seem to preserve in an individual a certain uniformity in their plan of arrangement. The investigation of the general or relative position of the various coils of the small intestine is a matter of much difficulty, and the plan of their disposition can be but very imperfectly inferred in the adult, from comparison with what obtains in the infant, where the scheme of arrangement is more apparent. The intestines move most freely in the upper left lateral and central regions of the abdomen, the coils in the former situation being mostly those of the jejunum, whilst the ileum chiefly occupies the right lower region. As a general rule the upper jejunal coils have a horizontal direction, as those of the ileum are mainly vertical (Addison, *loc. cit.*)

At a variable point in the course of the ileum there is frequently found on the free border a diverticulum (Meckel's) of 2 to 3 inches in length, of about the same calibre as the gut and formed of the same coats.

The **large intestine**, which reaches from the end of the ileum to the anus, is from 5 to 6 feet in length, and $2\frac{1}{2}$ inches in diameter, gradually decreasing towards the termination. Owing to a special arrangement of the muscular coat this portion of the alimentary canal is sacculated for the greater part of its length, and thus differs in appearance and in sectional area from the small bowel. It is separated into several parts, the anatomical relations of which are of considerable clinical importance.

The **cæcum** (*caput cæcum coli*) is the rounded blind end of the large intestine below the ileo-colic opening, of a length of $2\frac{1}{2}$ to 3 inches and similar diameter. It lies in the right iliac fossa upon the ileo-psoas muscle, the peritoneum, sub-peritoneal tissue, and iliac fascia intervening, and is in contact with the abdominal wall in front. The lowest point of the cæcum is generally in the right lateral line (*vide supra*), well below the level of the anterior superior iliac spine (Addison). It is usually invested by the peritoneum completely, and there is never any mesentery, but sometimes the serous membrane is reflected on to the posterior surface at a lower level, having the upper part of the cæcum uncovered

behind, which is then attached to the fascia by connective tissue continuous with that behind the ascending colon.

The cæcum occasionally occupies a position nearer to or even in the median plane, or higher up and closer to the liver, or it may have a wide range of movement and be found in the pelvis or even across the middle line.

Projecting from the inner and posterior aspect of the cæcum is the **vermiform appendix.** This is a narrow, slightly tapering prolongation of the bowel which in the fœtus is attached to the apex of the conical cæcum, but as development proceeds comes to occupy the position stated, as the end of the cæcum becomes rounded; sometimes (2 per cent, Treves) this infantile type persists. The average length of the appendix is 4 inches, and this may be attained in childhood; but it has been met with as short as 1 inch and as long as 9 inches. The diameter is half an inch.

The distal portion is entirely covered by peritoneum, the remainder being provided with a mesentery. Owing to this arrangement the appendix—unless very short, when it is straight—has a hooked shape, the extremity being bent on itself. In position it lies most frequently behind the lower end of the ileum, with a general direction to the left and upwards; in many cases its extremity hangs downwards into the pelvis, and then in the female it may come into close relation with the right ovary and Fallopian tube; less often it is directed upwards behind the cæcum and ascending colon, when it may be more or less concealed in a retro-colic pouch of peritoneum. The appendix opens into the cæcum about an inch below the ileo-colic valve, and the aperture, which is smaller than the rest of the lumen, is sometimes imperfectly valved. All degrees of obliteration of the cavity are met with, uniformly throughout or only in places.

The root of the appendix superficially corresponds to a spot about an inch below "Munro's point," which is rather internal to the junction of the middle and outer third of a line drawn from the anterior superior iliac spine to the umbilicus. This point marks the intersection of the spino-umbilical line and outer edge of the rectus. The point known as "M'Burney's" lies in the same line slightly external to Munro's, and between 1½ and 2 inches from the anterior spinous process of the "ileum."[1] Owing to the variable position of the appendix no surface indication can be laid down.

The terminal portion of the ileum, which lies in close apposition internal to and then behind the cæcum, opens into the posterior part of the large intestine by an almost transverse slit about ½ inch in length, protected by two lips, chiefly composed of mucous membrane with some fibres of the circular muscular coat, which project into the large bowel

[1] For anatomy of this region see paper by Sir F. Treves, Bart., *Brit. Med. Jl.*, 28th June 1902.

—the **ileo-colic valve.** This structure is sometimes only partly developed and occasionally is wanting. Its position is exactly marked on the surface by "Munro's point." From this it will be seen that whilst "M'Burney's point" is of some clinical value, "Munro's point" has precise anatomical significance.

The **ascending colon** extends from the cæcum to the under surface of the right lobe of the liver outside the gall-bladder, and is about 8 inches in length. It lies on the outer edge of the quadratus lumborum muscle and the lower part of the right kidney, being as a rule firmly attached by connective tissue to the lumbar fascia. The peritoneum passes over its sides and anterior surface without forming a mesentery; occasionally, however, a short meso-colon exists. A few coils of the ileum lie in front of the ascending colon, which is rather nearer the front of the abdomen than the descending portion is. A vertical line drawn upwards from the anterior iliac spine closely corresponds to the outer border of this section of the bowel.

At the level of the first lumbar vertebra, or of the 10th rib, in the mid-axillary line, the colon turns forwards and to the left, forming a bend known as the *hepatic flexure*, which is partly covered by the liver just within the costal margin and rather outside the right nipple line.

Passing from under the edge of the liver across the upper part of the umbilical region to the left hypochondrium is the **transverse colon**, forming an arch, or it may be several curves, with the convexity forwards and downwards. The level, however, at which this part of the intestine lies is very variable, and it may form so considerable a curve downwards that the lowest point reaches into the hypogastric region.

It is much the most movable part of the colon, and is furnished with a long transverse meso-colon, which, consisting of two layers of peritoneum directed forwards from the front of the pancreas and second part of the duodenum, completely invests the transverse colon, and meeting at the lower border passes downwards, thus forming the posterior layers of the great omentum, which intervenes between the transverse colon and the abdominal wall. Behind are the second part of the duodenum and coils of the jejunum; above it is in contact with the under surface of the right lobe of the liver, the gall-bladder, greater curvature of the stomach, and lower end of the spleen; and below are the coils of the small intestine.

The transverse colon ends by a sharp curve downwards—the *splenic flexure*—which is placed deeper in the abdomen than the hepatic curve, and at a rather higher level. From off this flexure to the diaphragm opposite the eleventh rib is a strong reflection of the peritoneum—the costo-colic ligament—passing between the colon and spleen and serving to support the latter.

The **descending colon** occupies a similar position on the left side to the ascending colon on the right, but is somewhat deeper from the

front of the abdomen and rather more external, lying in contact with the outer margin of the kidney; then, turning slightly inwards, it descends

NORMAL POSITION OF VISCERA BELOW THE DIAPHRAGM VIEWED FROM ABOVE (BRAUNE)

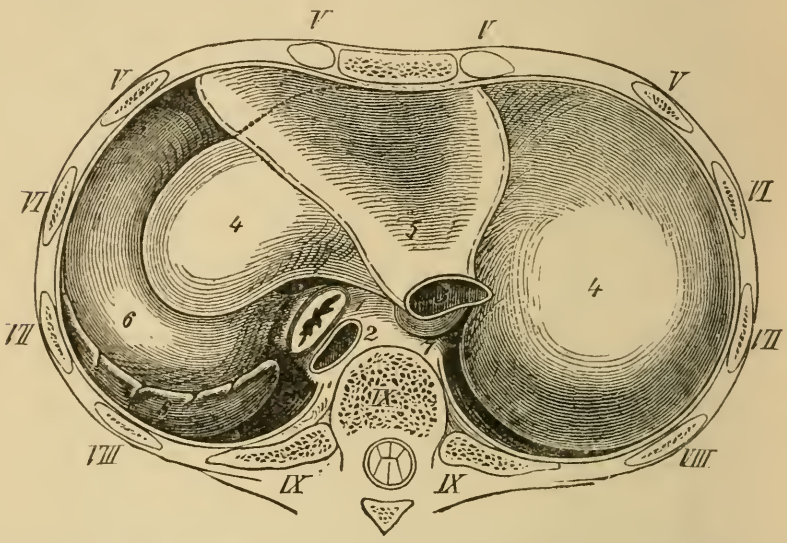

FIG. 2.—Shows the relation of parts, the stomach being moderately full. 1, œsophagus; 2, aorta; 3, inferior vena cava; 4, liver; 5, pericardial portion of diaphragm; 6, stomach; 7, lobulus spigelii; 8, spleen.

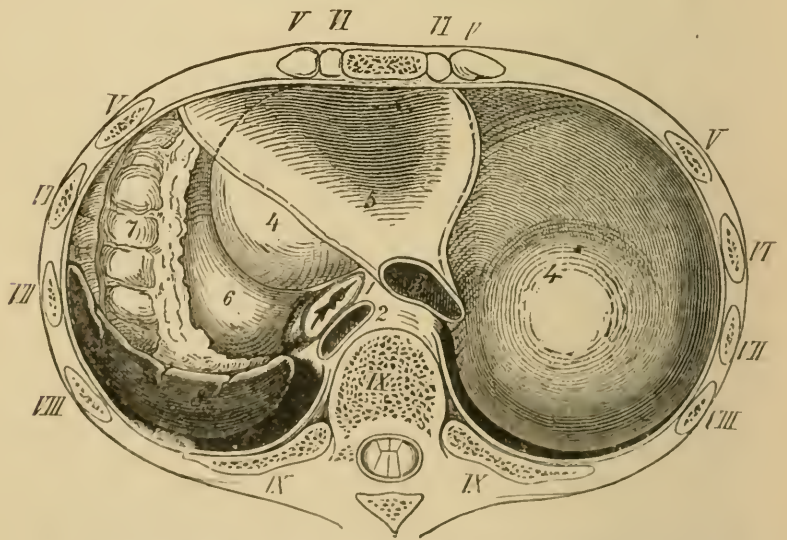

FIG. 3.—Stomach empty. Figures as above, except 7, splenic flexure of colon.

across the iliac fossa in its course to the pelvis—that part of the bowel from the iliac crest to the pelvic brim constituting the **sigmoid colon.**

The descending also like the ascending colon has no mesentery, being covered by peritoneum in its anterior and outer surfaces only, the remainder being attached by the post-peritoneal connective tissue. The first 2 or 3 inches of the sigmoid colon has a similar serous covering, being bound down on the iliac fascia in its course downwards and forwards, becoming more superficial close to the iliac spine. The lower part of this section has a distinct mesentery of several inches in length permitting of considerable mobility, and sometimes of sufficient length to allow this sigmoid flexure proper to pass down into the pelvis or upwards towards the umbilicus, or even higher. Both the descending and sigmoid colon are covered to a variable extent by coils of the small intestine.

The **rectum** continues from the sigmoid colon to the anal canal, and is about 8 inches in length. The upper half passes backwards and downwards from the pelvic brim to the middle line of the third sacral vertebra, and is furnished with a mesentery continuous with the meso-sigmoid. In front and to the sides lie coils of the ileum, with the ureter close to the left side. The lower section, which lies in the middle line of the sacrum and coccyx, has no mesentery, and is attached posteriorly by connective tissue, the peritoneum being reflected off the front and sides on to the bladder or uterus, leaving the greater part quite free from peritoneum and in contact in front with the base of the bladder, vesiculæ seminales, and prostate gland in the male, and with the pouch of Douglas and vagina in the female.

The **anal canal**, surrounded by the sphincters of the anus, extends from the rectum at almost a right angle down and backwards, and "measures fully an inch in length when the rectum is empty, but is shorter when the rectum is distended," forming "an antero-posterior slit in the pelvic floor, its lateral walls being in apposition," whereas "the lower part of the rectum when empty appears as a transverse slit" (Quain). Behind is the ano-coccygeal body, at the sides is the fat of the ischio-rectal fossæ, and in front the bulb of the corpus spongiosum in the male, and the perineal body which separates it from the end of the vagina in the female.

The relation of these viscera to the peritoneum is of considerable clinical importance, and is set forth on p. 483.

MINUTE STRUCTURE

The alimentary canal is lined throughout by mucous membrane variously disposed and with special characters in different situations. External to this is a coating of loose areolar tissue, with occasional fat cells—submucosa—in which the blood-vessels, lacteals, and nerves subdivide before their terminal distribution in the superficial layer.

Outside, again, is a muscular layer, also with some variation of arrangement in different parts. And external to all in the greater part of the canal contained in the abdomen is a serous covering derived from the peritoneum. Of these the mucous and muscular coats require special description.

It may be mentioned, as having some bearing upon the morbid changes occurring in these structures, that the epithelial lining of the mouth, except over the tongue, and of the salivary and mucous glands opening therein, is derived from the epiblastic infolding (stomodæum); similarly the anal epithelium originates from an infolding of the same layer (proctodæum); the rest of the alimentary epithelium, lingual, pharyngeal, œsophageal, gastric, and intestinal is derived from the hypoblast, including that of all the glands which open upon the mucous surface, pancreas, and liver. The whole of the muscular tissue, plain and striated, the connective tissue, the blood-vessels, and lymphatics, as well as the peritoneum, are throughout derived from the mesoblast. From the epiblast are formed the cerebro-spinal and sympathetic nerves.

MUCOUS MEMBRANE. — The surface is everywhere covered by *epithelium*, which in the mouth, pharynx, and gullet is of the squamous variety, arranged in a thick stratified layer, and is protective in function. This sharply changes at the cardiac orifice to a layer of columnar cells —averaging $\frac{1}{2000}$ inch in length—with which the stomach is lined, differing from those of the intestine in having no striated border; and when the surface is not secreting the cells are more granular in their deeper parts and clearer towards their outer zone, owing to mucigen which is discharged during digestion. In the duodenum the columnar epithelium at once assumes a striated free border, and maintains the same character throughout the small and large intestines. The cells are somewhat shorter than those of the stomach, but like them secrete mucus, and present after discharge of the same the peculiar "goblet-shape" appearance, the number of which varies considerably at different times. Lying among the tapering attached ends of the cells are numerous leucocytes, which are more numerous in the lower part of the ileum. The epithelium, which is essentially absorbent in function, is liable to contain abundant fat granules, which become black with osmic acid.

Throughout the stomach and intestines the epithelium is set upon a *basement membrane*, composed of a layer of flattened cells, closely connected with the branched cells of the retiform tissue, and from which the superimposed epithelium may be easily detached. At the anus there is a gradual transition of the epithelium into the epidermis. The deeper part of the mucosa is constructed of glands, blood-vessels, lymphatics, and nerves packed in a delicate stroma of areolar and adenoid tissues, among which are plain muscular fibre cells, which pass vertically upwards from the more or less definitely disposed layer

constituting the *muscularis mucosæ*, which separates the mucous from the submucous coat.

In the appendix the submucosa is intimately connected with the subperitoneal tissue by means of certain gaps in the muscular layers which serve for the transmission of blood-vessels, lymphatics, and nerves.

Throughout the whole length of the canal are myriads of microscopic *secreting glands* embedded in the mucous and submucous layers, and arranged for the most part vertically to the free surface upon which they open. They are involutions of the epithelial surface of a tubular, compound tubular, racemose, or tubulo-racemose type, the lining cells sometimes undergoing modifications in shape, tending in greater or less degree to be spheroidal—secretory—and are then for the most part concerned in the elaboration of a specific secretion, *e.g.* the gastric juice ; when the cells lining the gland retain the columnar form which clothes the general surface of the mucosa, the secretion is chiefly or entirely mucus.

The *buccal*, *lingual*, *pharyngeal*, and *œsophageal glands* are small and of the compound racemose type, more numerous at certain spots, and lined by columnar cells.

The *salivary glands* are of the compound racemose type. The alveoli, which open into the terminal ducts, consist of a fenestrated basement membrane, outside of which are the blood-vessels, lymphatics, and nerves, in a loose connective tissue structure, thus forming the lobules. The secreting cells which line the alveoli are of two kinds, some are large, rounded, clear from the accumulation within them of mucigen from which the mucin is formed, and almost fill the alveoli, the nuclei being situated nearer the attached end of the cells. The alveoli containing such cells and the glands (sublingual, labial, and buccal) made up of such alveoli are termed "mucous." In the parotid gland the alveolar cells are smaller, granular, and their nuclei are central in position ; these are termed "serous" glands. The submaxillary are "mixed" glands, composed of both mucous and serous alveoli, the latter being more numerous. In some of the mucous alveoli a third form of cell is to be found, the marginal cells or demilunes, which are small, very granular, lying next to the basement membrane, between that and the mucin cells.

The *gastric glands* are of the tubular variety, and are mostly, but not entirely subdivided towards their closed ends. Two kinds are to be recognised : (*a*) the pyloric glands, most numerous towards the pyloric end, open on the surface by wider mouths, the columnar epithelium being continued some distance down the tubule, forming the duct, the deeper portions being lined by shorter, more cubical, and finely granular nucleated cells ; (*b*) the cardiac or peptic glands, more abundant at the cardiac extremity, arranged in groups of four or five, and varying in length from $\frac{1}{20}$ to $\frac{1}{60}$ of an inch, are characterised by a shorter dis-

tance (duct), being lined by the columnar cells of the surface, beyond which the cells are like the distal cells of the pyloric glands, though more coarsely granular, and are known as the "principal" or "central" cells. Lying here and there among these, close to the basement membrane, are the parietal or "oxyntic" cells, which are much darker and more granular in the prepared specimen. The lumen of these glands is exceedingly fine, and still finer prolongations of it extend between the lining cells, distinct canalicular networks surrounding the parietal cells. There are also some tubules which are intermediate in character between these two forms.

The *intestinal glands* or *crypts of Lieberkühn* are distributed throughout the entire small and large intestines, though somewhat more abundantly in the latter, where they are probably solely mucus-producing. They are of the simple tubular pattern, of $\frac{1}{120}$ to $\frac{1}{300}$ inch in length, lined by columnar epithelium (the deepest cells of which, in the small intestine, are very granular), intermixed with goblet cells which are much more abundant in the large intestine. Just within the anus is "a narrow zone of mucous membrane destitute of glands" (Quain).

Brunner's glands are peculiar to the duodenum, in the first part of which they are most numerous. They are of the acino-tubular variety and are deeply placed in the submucosa, resembling in structure by gradual transition the pyloric glands of the stomach.

Isolated masses of the lymphoid tissue, of $\frac{1}{24}$ to $\frac{1}{12}$ inch in size, which is distributed throughout the mucous membranes and submucosa, the meshes of which are closely crowded with lymph-corpuscles, are met with sometimes in the stomach, constantly throughout the small and large intestines, chiefly in the ileum, cæcum, and appendix. These are known as the *solitary glands*, and are more or less distinctly marked off from the tissue in which they lie by their somewhat denser character, as well as by their surrounding vascular zone, fine branches of which permeate the nodule. Aggregations of these solitary glands, forming groups varying from $\frac{1}{2}$ an inch to 2 or more inches in length, and of $\frac{1}{2}$ an inch to 1 inch wide—the *agminated glands* or *Peyer's patches*—occur in the jejunum and ileum, increasing in number towards the ileo-colic valve. They lie lengthways in the intestine, mostly opposite to the line of the mesenteric attachment. These nodules, whether singly or in patches, extend from the submucous through the muscularis mucosæ into the mucous coat, and usually cause an appreciable prominence on the surface, especially in young persons, in whom they are larger, becoming atrophied as age advances.

The *tonsils* are oval masses of lymphoid tissue arranged in nodules or follicles, between which are tubular invaginations from the surface lined by stratified epithelium and known as crypts.

The surface of the mucous membrane of the alimentary canal is not uniformly smooth and even throughout, but is thrown into numer-

ous inequalities, some of which are temporary, disappearing almost or entirely as the part is fully distended, such as the rugæ of the gullet and stomach already described. Others form permanent extensions of the mucous surface, and thereby increase the secreting and absorbent area, as well as offering some slight obstruction to the progress of the intestinal contents, a delay which is physiologically desirable. The most important of these are the valvulæ conniventes, the villi, the saccules of the colon, and the plicæ or valves of the rectum.

The *valvulæ conniventes* are folds of the mucous membrane only, extending into the lumen of the bowel, usually crescentic in shape and encircling transversely a half to two-thirds of the circumference, occasionally forming complete circular projections, and sometimes exhibiting a tendency for a short distance to a spiral arrangement. Their greatest height seldom exceeds $\frac{1}{3}$ of an inch. They commence about 2 inches beyond the pylorus, are largest and most complete about the opening of the bile duct, and occur throughout the rest of the duodenum and upper half of the jejunum, beyond which they gradually become less and less distinct and disappear about the middle of the ileum.

From the duodenal side of the pyloric ring to the iliac aspect of the lips of the ileo-colic valve, the mucous membrane of the intestine is beset with innumerable fine projections—*villi*—the largest measuring $\frac{1}{50}$ to $\frac{1}{8}$ inch in length. They slightly decrease in size and number from the duodenum to the ileum, and thence onwards, and whilst broader and flatter above, become filiform lower down, and are for the most part wanting over the Peyer's patches. They are closely set and give to the surface a velvety appearance. The structure of a villus is that of the mucosa, of which it is but a simple projection. Covered by columnar epithelium and a basement membrane, each one contains a network of blood-vessels, one or more lacteals, which end either in a closed extremity or in a loop, and nerve fibrils, supported by loose retiform tissue, the branched cells of which are connected both with the walls of the lacteals and with the basement membrane, intermixed with a few muscular fibre cells, derived from the muscularis mucosæ, and specially arranged round the lacteal radicals. Rudimentary villi may be found scattered over the pyloric end of the stomach, but they are quite absent from the large intestine.

The colon is distinguished by being sacculated throughout its course, a condition that is due to a special arrangement of the longitudinal muscular layer to be presently described. The rectum is in some measure provided with similar *sacculations*, but this portion of the bowel is especially characterised by having its mucous membrane thrown into numerous transverse or oblique *folds* (*plicæ*) or "valves," which are most apparent in the empty and contracted state, a few only remaining when the gut is distended, and these, being of some size, offer a very real obstruction both to the passage of the contents and to the intro-

duction of the bougie, etc., from without. Within the anal canal and between it and the lowest permanent fold the rectum is more dilated than it is above. In the anal canal itself the mucous membrane forms several longitudinal folds on each side.

MUSCULAR COAT.—The various movements to which the food is submitted in its course through the alimentary canal are effected by special arrangements of muscular tissue placed external to the submucous layer. Except around the mouth, pharynx, and upper end of the œsophagus, and the external sphincter of the anus, when it is of the striated, voluntary character, the coat is composed of plain, involuntary fibre cells disposed in an inner circular and outer longitudinal layer, with modifications at certain situations, the former being generally the thicker.

The muscles which enter into the formation of the walls of the *buccal cavity* are those which move the lower jaw, and those which form the lips, cheeks, and floor of the mouth. The former group, consisting of the masseters, temporals, and pterygoids, supplied by the fifth cranial nerve, are attached above to the superior maxilla, sphenoid, and temporal bones, and are inserted into the lower jaw at different angles. The other groups comprise the orbicularis oris, elevators and depressors of the lips and angles of the mouth, the zygomatici and buccinators, supplied by the seventh cranial nerve, and the muscles of the tongue, supplied by the hypoglossal nerves.

The muscular wall of the *pharynx* consists of fibres which are separable into three more or less distinct portions, named the upper, middle, and lower constrictors, attached on each side in front to the bones, ligaments, mucous membrane, and cartilages which intervene between the internal pterygoid plate of the sphenoid and the cricoid cartilage; passing backwards they spread out, and meet in the middle line posteriorly, where they form a continuous layer. Internal to this coat there are on each side two special muscular bundles, the stylo-pharyngeus and palato-pharyngeus, which, arising above from the styloid process and soft palate respectively, pass downwards and blend with the constrictors with some attachment to the thyroid cartilage. The nerve-supply of this part of the alimentary canal is derived from the spinal accessory, glosso-pharyngeal, and laryngeal branches of the vagus.

In the *œsophagus* the longitudinal fibres which are connected with the lower pharyngeal constrictor start from a common tendon, which is attached to the back of the cricoid cartilage, and, spreading backwards and downwards, come to form a complete layer over the tube; the circular fibres which above fill in the interval between the longitudinal bundles also blend with the inferior constrictor and form a continuous series of rings down to the stomach, the two layers together forming a thicker coat than is found elsewhere in the canal. Bands of plain

muscular tissue are frequently found, passing from the longitudinal layer to the left pleura or bronchus, or to both.

The muscular coat of the *stomach* consists of several layers of plain fibre cells arranged somewhat differently at different parts of the organ. Most externally the fibres are disposed longitudinally, and continuous with those of the gullet pass chiefly along the curvatures of the stomach, being much less distinct over the surfaces, to the plyorus, where they blend with the corresponding fibres of the duodenum. The fibres of the middle layer are mostly circular in direction, forming a complete investment of the organ from fundus to pylorus, where a specially thickened bundle forms the pyloric sphincter. The most internal fibres are distributed obliquely over the surfaces of the stomach, and whilst well marked over the fundus scarcely reach the pyloric end.

Uniformly throughout the *small intestine* the musculature is disposed in the ordinary two layers, becoming gradually thinner towards the lower end. No extension of this coat takes place into the valvulæ conniventes.

A similar arrangement obtains in the vermiform appendix, although there is greater irregularity in distribution of the fibres, and at places considerable gaps are to be found. Elsewhere in the entire course of the *colon*, and of all but the lowest part of the rectum, the two muscular layers are but very sparsely distributed over the bowel, being specially collected into definite muscular bundles. Those of the outer layer form three bands, which, starting from the root of the appendix, traverse the cæcum, colon, sigmoid flexure, and rectum, one posteriorly along the attached aspect; another along the anterior border corresponding in the transverse colon to the reflection of the great omentum; and a third on the inner aspect of the ascending and descending colon and under side of the transverse portion. Inasmuch as these bands are considerably shorter than the large intestine itself, the latter is necessarily gathered up into sacculi in order to be accommodated within their length. There is thus produced externally a series of constrictions corresponding to sharp projecting folds seen in the interior of the bowel; at these constrictions the circular fibres are mostly collected. Some of the circular fibres, with those from the ileum, enter into the formation of the lips of the ileo-colic valve, but the longitudinal fibres are continued on without taking any part in their structure.

In the *rectum* the longitudinal fibres are arranged chiefly in bands on the anterior and posterior surfaces, and the circular, as those in the colon. In the *anal canal* the circular are represented by a thick bundle —the internal sphincter—external to which is a continuation of the longitudinal fibres, which are joined by special bands of plain muscular tissue passing down and forward from the coccyx. Encircling the anus is a flattened layer of striated muscle—the external sphincter—attached posteriorly to the coccyx, and in front to the central point of the perinæum, and to the skin. Connected also with the rectum are the

anterior portions of the levatores ani, which pass down and inwards from their attachment to the inner aspect of the pubes and pelvic fascia, to be inserted partly into the wall of the rectum, blending with the longitudinal fibres, and partly join their fellows in the middle line and the tip of the coccyx.

The SEROUS COVERING, which allows by its elasticity the varying distension of the organs, is an exceedingly thin, homogeneous basement membrane upon which is a layer of flattened endothelium, and connected by areolar tissue with the subjacent coat. Where the peritoneum is wanting the areolar tissue becomes continuous with the post-peritoneal connective tissue.

VASCULAR SUPPLY

The œsophagus is supplied by small branches from the descending thoracic aorta, the inferior thyroid, left inferior phrenic, and coronary of the stomach which anastomose, the return being effected by corresponding veins, which reach the superior cava *viâ* the azygos. Thus an arterial and venous communication is established with the arteries of the abdominal part of the alimentary canal and with the radicals of the portal vein. The source and distribution of the blood-vessels of the gastro-intestinal tract are detailed on table opposite p. 523.

Within the thickness of the walls of the canal the arteries give off branches which form capillary networks within the several coats, but especially around the glands and in the villi of the mucosa. These branches reach the stomach and intestines either between the layers of the mesenteries, or, where these do not exist, by the post-peritoneal tissue.

LYMPHATIC VESSELS

The lymphatic vessels commence in lymph spaces in the mucosa among the glands and blood-vessels. Thence they pass through the muscularis mucosæ, and form plexuses in the submucosa, whence large branches pass through to the surface of the organs, and finally reach the thoracic duct, passing in their course through various lymphatic glands, deep cervical, posterior mediastinal, mesenteric, and retroperitoneal. In the small intestine additional plexuses of lacteals lie between the layers of the muscular coat, as well as among the fibres, and a further set are arranged beneath the serous coats. These several networks all freely communicate. In the villi the lacteal radicals are found as single vessels, with bulbous closed ends, or as simple loops.

The lymph spaces and vessels are closely connected with the lymphoid nodules, both solitary and agminated, forming special networks around them.

THE NERVOUS SUPPLY

The nerves to the mouth and pharynx have been mentioned in connection with the muscles of these regions.

The œsophagus is supplied by twigs from the trunks and recurrent laryngeal branches of the vagi, from the glosso-pharyngeal, and from the sympathetic, which end in gangliated plexuses between the muscular layers and in the submucosa, from which terminal filaments are distributed to the muscle fibres, glands, and between the epithelial cells.

The nerves of the stomach originate from both pneumogastrics, the terminal branches of the left being distributed over the anterior surface, and those of the right over the posterior surface, chiefly towards the pyloric end. Branches of the sympathetic system from the solar plexus also reach the stomach. The small intestine is supplied by branches from the superior mesenteric plexus, which is formed from the sympathetic and vagus. A similar arrangement obtains in the colon, where filaments from the lower dorsal, upper lumbar, and second and third sacral nerves correspond to the vagal fibres to the stomach and small intestines. From all these sources branches pass through the walls of the stomach and intestine to communicate with gangliated plexuses lying between the muscular layers (Auerbach) and in the submucosa (Meissner). Filaments from these supply the muscular tissue and the mucosa.

Thus the efferent and afferent channels to and from the viscera (stomach and intestines) and the nerve centres lie in the vagi, and in the white rami communicantes of the sympathetic from the sixth to the tenth dorsal spinal segments.

The Physiology of Digestion

The larger proportion of the solid ingesta—proteids, fats, and carbohydrates—are, as they are presented to the digestive organs, capable of being but very slightly absorbed into the blood current, which is an indispensable preliminary to their distribution to the tissues. It becomes necessary, therefore, that they should be converted to an absorbable condition, retaining meanwhile their separate characteristics. The physico-chemical processes by which these changes are effected, together with certain muscular mechanisms by which the foods are subdivided, masticated, swallowed, mixed, and propelled along the alimentary canal, constitute the function of digestion. The rest of the ingesta, such as the water and the soluble mineral substances, except so far as these last may require separation from the proteid and other bodies with which they may be associated, require, like the oxygen in respiration, no

previous preparation to fit them for absorption from the surfaces of the gastro-intestinal mucous membrane.

It is most desirable that the conception of "digestion" be strictly limited to the process as thus defined, and that it should not be taken to include the function of "absorption." The latter is wholly different in its nature from the complex chemical changes which constitute the former, and are capable of almost an exact imitation outside the body. Absorption is the second stage in the progress of the ingested food-stuffs towards the nutrition of the tissues, when for the first time the digesta are brought within the range of living protoplasmic activity, as they traverse the columnar epithelial cells of the intestinal mucosa. In the blood, in the liver, and ultimately in the tissue elements themselves, the absorbed nutritive material undergoes still further metabolism, which so far is incapable of imitation in the laboratory, and is inseparably associated with the presence of living matter. A sharp line may, therefore, be drawn between digestion and the subsequent changes in its products, and this line justified physiologically should be observed pathologically.

Further, it may be well to recognise that the digestive changes in the food are a natural and more elaborate continuance of those artificial processes, such as cooking, to which much of the aliment is subjected, and that although digestion is carried out in the course of the alimentary canal, that tract may not improperly be regarded as outside the blood current and the tissues of the body as much as the skin is, forming as it does a highly specialised extension of the surface.

Clearly the first consideration in respect to digestion is the **food**, its nature and character, more particularly the quantity and its apportionment into properly regulated meals at suitable intervals; the state of mechanical subdivision as effected by cooking, mastication, etc., in which it is presented to the digestive juices; its freedom or otherwise from physically injurious or poisonous constituents; and, lastly, its nutritive composition, that is, the needful proportion of the several alimentary principles. It may be observed that this last character is not synonymous with "digestibility" as too often supposed; many very nutritive substances requiring considerable digestion to fit them for absorption, whilst others of no nutritive value, or even harmful, are readily absorbed, making little or no demand on the digestive process. With the consideration of food and diet the next section is specially concerned.

The digestive secretions.—The solution and conversion into an absorbable state of the several alimentary principles is carried out by the secretions of the salivary, gastric, and intestinal glands and of the pancreas. Several pints of these fluids, taken collectively, are poured out into the alimentary canal daily, the greater part being reabsorbed. They are in some measure aided by the water taken with the food, a certain quantity of which is needed for enabling a due mixture of the gastro-intestinal

contents, as an excessive amount may be detrimental by diluting the digestive juices.

The efficiency of these secretions depends upon the presence in them of special ferments or enzymes, which are the products of the metabolic activity of the gland cells. To a similar source the hydrochloric acid of the gastric juice is to be attributed, the parietal cells of the peptic glands being regarded as specially concerned in its formation from the chlorides of the food, probably by the action thereon of the alkaline phosphates. The exact mode of action of these ferment bodies is far from being satisfactorily known; an exceedingly minute quantity suffices to effect the specific changes in bringing about which they appear to suffer no loss. Short of precipitation their activity seems to be of indefinite duration, and they are unaffected by such substances as antiseptics, which are poisonous to the living cells in which they are formed. The change they determine in the several alimentary principles is of the nature of hydrolysis, the substance acted on taking up the elements of water. For their satisfactory action certain conditions must exist, such as a temperature corresponding to that of the body (at a heat of 60° to 65° C. they are permanently destroyed), and a fairly constant state of acidity or alkalinity of the medium according to the special secretion; thus the *pepsin* and *rennin* formed by the central cells of the gastric glands act best in the presence of 0.2 to 0.3 per cent of hydrochloric acid, a higher percentage than 0.4 hindering the action of the former, which is stopped altogether in an alkaline solution. On the other hand, the pancreatic enzymes, trypsin, amylopsin, and steapsin, will only act in an alkaline medium, which is produced by the sodium carbonate of the secretion (as well as the alkaline bile), their efficacy being destroyed in the presence of an acid. The *ptyalin* of the saliva also requires a neutral or slightly alkaline reaction, any acidity averting its diastatic power. The intestinal juice is also alkaline in reaction. Most important is it that the products of their digestion should be absorbed out of the way, as an accumulation of their results seriously impedes or altogether stops their further activity.

The several secretions, which range in sp. gr. from 1001 to 1010 of the gastric, to 1030 of the pancreatic, contain different salts, of which the most abundant are the chlorides of potassium and sodium, which occur in all, sodium carbonate in the saliva, pancreatic and intestinal juices, small quantities of sodium phosphate in the saliva and gastric and pancreatic juices, and occasional traces of potassium sulphocyanate in the saliva.

The bile plays a very subordinate part in digestion, chiefly aiding the pancreatic juice in virtue of its alkalinity, and as a stimulus to the intestinal movements. As an antiseptic bile seems to be of doubtful efficacy. It is largely of an excretory character, and will be further considered in connection with diseases of the liver.

Throughout the whole length of the canal, but more especially in the mouth, stomach, and colon, a very large quantity of *mucus* is formed. This secretion exerts little, if any, digestive action, though some inverting action on cane sugar is claimed for it, but it serves an important use in lubricating the food in its passage over the delicate mucous surface.

The secretory activity of the various glands is distinctly controlled by the nervous system, and the precise anatomical distribution of the nerves concerned is in some cases known. Thus the nerves of the salivary glands are derived from the cervical sympathetic, and from the chorda tympani of the facial, and stimulation of each separately causes the secretion of a saliva varying in character and action. The gastric glands and the pancreas are controlled by the vagi and the sympathetic nerves, along both of which pass impulses that excite the secretion of water, acid, salts, and ferments in varying proportions, according in great measure to the nature of the food. It further appears that the stomach and pancreas are liable to be inhibited in their secretory activity by impressions that reach them *viâ* the pneumogastrics.

The normal stimulus to the salivary secretion has long been known to be of a psychic character induced by the sight, smell, or thought of food, together with the special excitation which certain sapid articles possess. The mere dryness of the ingesta also largely determines the quantity secreted. The elaborate experimental researches of Prof. Pawlow[1] and his co-workers have shown that by far the most potent excitant of the gastric secretion is the normal appetite. In the absence of a healthy desire for food the gastric juice is most insufficiently produced, but when this is present the flow commences about five minutes after the food enters the stomach (during which latent period the amylolytic action of the saliva continues), and goes on for several hours, gradually diminishing in quantity. Even more important are the results which these observers have obtained in respect to the influence excited on the secretion of the digestive fluids by different kinds of food. Briefly stated, it seems that the quantity and quality (*i.e.* the proportions of water, acid, ferment, etc.) of any juice is determined by the nature of the aliment, and in this way each different kind of food excites the secretion of a juice best fitted for its own digestion, richer or poorer in acid or ferment, and in greater or less quantity as may be required. Thus the secretion that follows on a meat diet differs in amount and composition from that excited by bread, as this again differs from what ensues on taking milk. In this way the gastric juice poured out as the result of normal hunger comes to be modified according to the food taken, and Prof. Pawlow maintains that this food-induced secretion is brought about by reflex excitation, due to a specific stimulation of the

[1] *The Work of the Digestive Glands*, by Prof. Pawlow. Translated by Prof. Thompson, 1902.

terminals of the afferent nerves in the mucous membrane by certain chemical qualities of the food. Meat extracts of various kinds are the most effective in promoting a flow of gastric juice, and there is good reason to believe that starch foods when combined with proteids increase the formation of pepsin. Fats, on the contrary, distinctly prevent the stomach secretion, and an accumulation of hydrochloric acid in the organ acts in the same way. Similar though not identical results have been obtained in respect to the pancreatic juice. Whilst the appetite excites but little influence on its secretion, acids of any kind, such as the chyme, act as powerful excitants of the flow. Here also different foods differently affect the secretion ; meat extracts have but a very slight effect, fats cause an increase, and starches possibly induce a large formation of amylolytic ferment. The efficacy and rapidity of action of the pancreatic juice are considerably augmented by the bile and succus entericus (which are also influenced in quantity and quality by the nature of the food), the former, according to Pawlow, specially exciting the production of the fat-splitting ferment, as the latter calls forth a pancreatic juice richer in proteolytic enzyme. The great importance of all this in the establishment of digestive habits and capabilities is obvious.

The digestion of the various alimentary principles—i. *Proteids.*—In whatever form these are presented to the digestive organs, whether albumins, globulins, fibrin, etc., they are converted into a fluid and diffusible condition by the pepsin of the gastric juice acting in association with hydrochloric acid and by the trypsin of the pancreatic secretion in the presence of an alkali. The final products of the action of these agents is essentially the formation of diffusible proteids—*peptones*, which result equally from peptic as from tryptic digestion, though important differences exist between the two processes. Thus, in the stomach, after the proteids have been converted into syntonin or acid albumin, various proteoses or albumoses occur, as well as peptones, whilst in the intestine but little proteose is formed from such proteids as have escaped gastric digestion. The trypsin also effects a further change than is brought about by the pepsin, for the peptones, which represent the terminal stage in the stomach, are decomposed in the bowel into various nitrogenous bodies, of which the amido-acids (leucin and tyrosin) are the most abundant. It is probable, however, that in normal conditions the tryptic digestion only proceeds to these final stages to a very limited extent. It would certainly seem that the pancreatic juice is capable of effecting a satisfactory digestion of the proteid food without any previous change therein by the gastric secretion, than which it acts much more rapidly. At least this can be experimentally shown outside the body; and in the remarkable cases in the human subject where the stomach has been completely resected for disease, within a few weeks after the operation the patient has been

able to take without discomfort an ordinary diet, consisting of meat, bread, eggs, milk, etc., and an investigation of the excreta as compared with the ingesta demonstrated a state of complete nitrogenous equilibrium, the patient gaining weight. Inasmuch, however, as but little absorption of the digesta takes place in the stomach, and as the proteids of the food are in the main submitted to pancreatic digestion in the form of albumoses and peptones, it is to be supposed that in the healthy organism the changes effected by the trypsin are more economically and suitably performed upon these substances, than on the crude proteids of the food. One of the principal of these, viz. the caseinogen of milk, is coagulated by a special enzyme—rennin—which is secreted like pepsin in the glands of the fundus of the stomach, the curds or casein being subsequently digested like other proteids. This change, though resembling the curdling of milk on the addition of acids, is not identical, and the presence of calcium salts, such as naturally occur in milk, is essential, just as in the coagulation of the blood.

Various "compound proteids" form no inconsiderable part of the nitrogenous intake, such as hæmoglobin and its compounds; glucoproteids, compounds of proteids with carbohydrates, as mucin; nuclein, compounds of proteids with phosphoric acid, found in egg-yolk, brain, etc.; nucleo-proteids, compounds of proteid with nuclein, found in the nuclei and protoplasm of cells; and lecith-albumins, compounds of proteids with lecithin, found in liver, kidney, spleen, etc. These substances are first split up by digestion into the proteid molecule and the organic radicle, the former being digested in the ordinary way, the latter often remaining unaffected. The group of proteids known as albuminoids, such as elastin, collagen, gelatin, keratin, lardacein, etc., offer considerable resistance to the action of the digestive juices, although some are split up into bodies closely resembling the peptones of proteid digestion. The most important of these bodies from a nutritive point of view is gelatin, which, though it cannot wholly replace proteid as an article of diet, is nevertheless a valuable proteid-sparer subsequent to its digestion and absorption. It is to be noted that collagen, which is the chief constituent of the connective tissues, cannot be digested by trypsin, though it is by pepsin and hydrochloric acid.

ii. *Carbohydrates.*—The changes which take place in these bodies in the digestive tract are less complex than those which the proteids undergo. The amyloses or starches are mainly converted by the ptyalin and amylopsin into maltose, which is a disaccharide ($C^{12}H^{22}O^{11}$), small quantities of grape sugar (glucose, dextrose) being coincidently formed. But it is not as maltose that the digested starches are finally absorbed; it as well as the soluble saccharoses (cane sugar, sugar of milk, etc.) of the food are found in the blood as dextrose, a monosaccharide ($C^6H^{12}O^6$), the inversion of the di- to the mono-saccharide being effected partly by the hydrochloric acid of the gastric juice and

partly by a special inverting ferment contained in the intestinal secretion. Some part also of this conversion probably takes place during the actual process of absorption. Associated with the digestive changes of the carbohydrates in the stomach is the formation of some lactic, with traces of acetic and butyric acids.

iii. *Fats.*—The neutral fats of the food are mostly unaffected until they come within reach of the pancreatic juice in the intestine, though when enveloped in a proteid covering, as in the cells of adipose tissue, or in milk, this is digested off and the fat liberated. The action of the pancreatic ferment—steapsin—consists in splitting the fats into their constituent fatty acids and glycerin, a change which is much facilitated by the presence of the bile salts; some of the acids so produced combine with alkalies in the intestine and form soaps. Probably the greater part of the fats taken are treated in this way. The presence of the free fatty acids and of alkalies, however, is found to favour the conversion of such fat as has not been decomposed into an emulsion, *i.e.* an extreme state of subdivision, the particles remaining separate.

The mutual interference and assistance of the several alimentary constituents in the course of their digestion are circumstances of considerable importance, and to be taken into account in the treatment of dyspepsia and the framing of a suitable dietary. And still more is this so in respect to certain food accessories, such as alcohol, tea, coffee, and condiments. The first-named, unless very concentrated, appears to be less harmful to digestion than is ordinarily supposed, and being one of the few materials which are absorbed from the stomach, its stimulant effect on the circulation may be actually beneficial to the digestive process, especially in a condition of exhaustion. Experimentally, it has been shown that tea and coffee do interfere with the peptic digestion of proteids. Further information on this subject is much to be desired.

The process of digestion consists, therefore, of a continuous series of physico-chemical changes commencing in the mouth with the action of the saliva on the masticated starch foods, which is continued in the stomach during the first part of the stay of the aliment in that organ, and until the hydrochloric acid has been secreted in sufficient quantity to more than neutralise the alkalinity of the meal. Hence a considerable share of the salivary digestion occurs in the stomach. In the stomach there is required a sufficiency of gastric juice containing neither an excess nor deficiency of hydrochloric acid (.1 to .25 per cent) poured out when food is there, and not when the organ is empty. The acid partly combines with the proteids present and in part (not more than 1 per 1000) remains free. By this agent, combined with pepsin, the nitrogenous alimentary principles are chiefly digested and passed on into the intestines as albumoses and peptones; the caseinogen of milk is coagulated by rennin, and converted into casein, which is afterwards

peptonised. From this it follows that an excess of meat food in a meal gives a better chance to the continued digestion of the farinaceous food in the stomach by using up the acid, whilst an excess of starches or sugars diminishes the opportunity of their conversion. Further, the normal lactic, acetic, and other bacterial fermentations which take place in the carbohydrate food-stuffs partially increase after the middle period of gastric digestion, but are normally held in check by the free hydrochloric acid present, whilst a deficiency of this material or a great excess of carbohydrates leads to the formation of undue quantities of the organic acids. The neutral fats of the food ordinarily undergo but little change beyond being melted in the stomach, only a slight decomposition and liberation of fatty acids, possibly due to bacterial agency. Hence portions of meat or other substances, that might be satisfactorily dealt with in the mouth or stomach, may pass on undigested if coated with a layer of fat such as melted butter. The various salts, phosphates, carbonates, malates, and other inorganic compounds occurring in the food, undergo some decomposition by the hydrochloric acid. Excessive quantities of some of these substances are apt prejudicially to affect the activity of the gastric secretion.

The period of time required for the completion of gastric digestion will necessarily vary with the bulk and nature of the meal, but as a general rule may be reckoned at three to five hours. The rate of action of the several ferments is variable : that of ptyalin and rennin is rapid, whilst pepsin differs much with different proteids, those of animal origin being generally more rapidly converted than the vegetable ; among the former casein and fibrin are quickly peptonised, whilst egg-albumin requires a very considerable time. Previous proper cooking of the food facilitates its separation, and to that extent the action of the secretions, but its effect on proteids does not in all cases render them more susceptible to ferment change.

Regarded as a whole, the digestive changes which take place in the stomach may be looked upon as a preliminary—desirable, but not essential—to those which are carried on lower down in the intestine. The acid chyme as it passes through the pylorus contains sugars (maltose, dextrose, etc.), proteoses and peptones, fatty acids and glycerin, together with water, salts, the constituents of the gastric juice, and such substances as have as yet escaped any digestive action, and indigestible residue, as well as micro-organisms. During the very short time occupied by the passage of the chyme through the first half of the duodenum nothing special is to be noted ; but below the entrance of the biliary and pancreatic ducts the alkaline fluids then poured into the canal neutralise the acidity of the chyme and cause a precipitation of albumoses, acid albumin, and the bile acids, with arrest of peptic action. The way is also cleared for a further digestion of the proteids, and of the starches and of the fats, by the pancreatic juice, the several ferments

of which, there is some reason to believe, act even more efficiently than do those of the gastric juice and saliva, and quite as rapidly, and, as seems possible, can do so without such assistance and preparation as may be afforded in the mouth and stomach. They are, however, materially aided in their action by the bile, which, possessing no special ferment of its own, acts by its alkalinity, and also by means of the biliary salts. The rate of progress of the intestinal contents, and the changes which take place therein, depend in some measure upon the quantity and nature of the food. How far the succus entericus is effective in digestive action in the human subject is uncertain ; its action on cane sugar has been referred to, and it is further claimed to assist, by means of a special ferment, in peptonising proteids. From pylorus to rectum the contents may be reckoned as occupying about eight hours in their passage, most of that time being spent in the colon.

Normally associated with the digestive process in the stomach and the intestines is a variable quantity of several *gases*. Some of these, such as oxygen and nitrogen, which particularly occur in the stomach, are swallowed with the food and saliva. Others, as carbonic acid, are said to be transferred from the blood. And a third set, carbonic acid, hydrogen, carburetted and sulphuretted hydrogen, are the products of fermentation, especially of carbohydrate and other vegetable food. No definite statement can be made as to when the quantities of these gases become excessive.

Within a few days after birth **micro-organisms** of various kinds (p. 53) are to be found in all parts of the alimentary canal—mouth, stomach, and intestines—and they are never afterwards absent, though very variable in number and kind, according as the state of the digestive process is favourable or otherwise to their development. Although undoubtedly aiding in the decomposition of the gastro-intestinal contents, and sometimes in a favourable manner, their presence cannot be regarded as an essential of the digestive process, and they very largely add to the risk of morbid action. In the mouth and also in the stomach they play but little part, the hydrochloric acid being a powerful germicide, any effect they have being connected with the lactic and butyric acid fermentation of the carbohydrates, and possibly with some splitting up of the fats. In the small intestine, however, and still more in the large, they very distinctly contribute to the changes which take place. It is the carbohydrates, both sugars and starches, which under normal conditions are most affected, various acids, such as lactic, butyric, and succinic being produced, which gradually alter the alkaline reaction of the duodenal contents as they pass along the lower lengths of the bowel. Traces of ethylic alcohol are also formed together with hydrogen and carbonic acid. It is supposed that some of the cellulose contained in the vegetable food, which resists the action of the digestive secretions, undergoes a splitting up by some of the intestinal bacteria,

but this is probably to only a very limited extent. "The changes brought about in the intestine by the action of bacteria on proteids are very similar to, and probably identical with, those which occur when proteids undergo putrefaction in the air, with this important exception, that those putrefactive bacteria, which produce the class of poisonous nitrogenous (alkaloidal) bases known as ptomaines, do not grow under normal conditions in the intestine. . . . Ptomaines, and especially poisonous ones, are found only in the later stages of putrefaction. It has also been suggested that the cause may be the absence of oxygen from the intestines, the ptomaine-forming bacteria being aërobic" (B. Moore, M.A., "The Chemistry of the Digestive Processes," in Prof. Schäfer's *Text-book of Physiology*, 1898). The changes at first resemble those brought about by trypsin, but they proceed much beyond, the resulting tyrosin being further split up into various members of the aromatic series, whilst such substances as indol and skatol, to which the odour of the fæces is due, are produced chiefly from the decomposition of the proteids (albumoses, peptones, etc.) without the previous formation of tyrosin. In addition, gases such as hydrogen and sulphuretted hydrogen are liberated. In the course of normal intestinal digestion the fats are but little acted on by organisms in the canal.

The movements of digestion.—By means of various arrangements of the muscular tissue, which lies, as already described, outside the mucous membrane of the alimentary canal, are performed the several movements necessary for the mastication, swallowing, and mixing of the ingesta, as well as their propulsion over the many feet of absorbent surface presented by the intestine, and, finally, the extrusion of the indigestible residue. The muscular tissue concerned in the movements of the terminal sections of the canal is of the striated variety, and the acts are more or less completely voluntary, being controlled almost entirely by branches of the cerebrospinal nervous system. The movements of the gullet, stomach, and intestines, on the contrary, are quite involuntary and unconsciously performed by plain muscular fibres, governed by a nerve-supply derived from the sympathetic system.

The movements are much facilitated by the natural fluidity of the food, which is further aided by admixture with the digestive secretions, and especially the mucus, by which the whole tract is lubricated.

Mastication.—The requisite subdivision of the solid food is effected by the cutting and grinding action of the incisor and molar teeth, which are brought into action by the movements of the lower jaw, in various directions, up and down, from side to side, and to and fro, by means of the temporal, masseter, pterygoid, digastric, genio- and mylo-hyloid muscles. The successive portions of material are kept between the teeth by the tongue and cheek muscles—buccinators. The sense impressions by which the co-ordination of the act of mastication is guided travel centripetally by branches of the fifth cranial nerve, whilst the

motor impulses reach the muscles of the jaw *viâ* the motor branch of the same nerve, those to the tongue by the twelfth, and by the seventh to the digastric and muscles of the cheeks and lips.

The portion of masticated food is then passed from the mouth into the stomach by *swallowing*, which it is usual to describe as consisting of three steps. The first consists in the compression from before back of the tongue against the hard palate, the teeth being closed, by which the material is squeezed into the fauces. This stage is wholly voluntary and effected by impulses which travel by the facial and hypoglossal nerves. In the second stage the food is forced through the pharynx, the soft palate being raised, and so, with the contracting palato-pharyngei shutting off the nasal region, at the same time the glottis is closed by its muscles, and the larynx is drawn up by the thyro-hyoid muscles against the epiglottis and base of the tongue ; the coincident contraction of the other elevators of the pharynx—the stylo-pharyngei—draws this structure up and over the mass of food, which thus comes within the grasp of the constrictors, by which it is propelled into the œsophagus. This stage of the act is mainly reflex and is the most rapidly performed; the afferent nerves are branches of the fifth cranial and superior laryngeal of vagus, and the efferent are the glosso-pharyngeal, vagus, and seventh cranial. The passage of the material along the œsophagus, or third stage of deglutition, is also reflex in character, and is in the main involuntary, though there are great individual variations in the extent to which it is controllable by the will. The reflex nervous mechanism depends upon impulses chiefly conducted along the fibres—centripetal and centrifugal—by branches of the vagus. The glosso-pharyngeal nerve conveys afferent impulses inhibiting œsophageal peristalsis, and so preventing the interference of waves resulting from successive acts of swallowing. It has been shown that the peristaltic wave does not spread by continuity along the musculature of the tube, but depends upon successive waves of vagal impulses ; its passage is hence not stopped by lesions of continuity of the gullet. During inspiration the muscular fibres of the diaphragm close the tube.

The *movements of the stomach* are of two kinds : in the cardiac two-thirds of the organ, when the acid is secreted and digestion takes place, the contents are submitted to a churning action whereby they are mixed with the secretion. When the material is reduced to a sufficiently fluid condition, it is passed on through the pre-pyloric sphincter into the pyloric region by more or less rhythmically propulsive efforts, by which it is propelled through the relaxed pyloric orifice. It would also seem that the antrum pylori can reject and force back towards the fundus such solid masses as may have escaped solution. These movements of the stomach are mainly stimulated by the character of the food, warm and hot ingesta specially exciting them ; and though over-acidity calls forth the peristalsis, it appears to induce a spasmodic and even painful spasm

of the pylorus with consequent hindrance to the escape of the chyme. Normally, the passage through the pylorus of the fluid foods, unless requiring gastric digestion, begins to take place as soon as swallowed.

Intestinal movements.—The experimental observation of the intestines shows that they are possessed of two forms of movements. The one, termed myogenic, consists of rhythmic swayings of the coils due to rapid waves of contraction involving both muscular layers at the same time, and are manifested when all connection with the central nervous system is severed. These movements are ineffectual for the propulsion of the contents, and probably serve the purpose of mixing them and bringing them in contact with the absorbing surface. Besides these automatic movements is the true peristalsis, by which the food is forced onwards, and are slower than the former. "The passage of the bolus downwards is rendered possible by an inhibition of all the spontaneous contractions of the intestinal wall for some distance below the bolus, so that the advancing wave drives the bolus always into a relaxed portion of the gut. A peristaltic wave, therefore, is a co-ordinated act involving two factors, an excitation above and an inhibition below the excited spot. . . . Just as at the upper end of the alimentary canal there is a gradual transition from the co-ordinated cerebrospinal reflex of deglutition to the local automatic movements in the stomach, so in the large intestine we find a gradual transition in the opposite direction, the activity of the gut becoming less automatic and more dependent in its extrinsic innervation as we proceed from ileo-colic valve to anus" (Starling). The myogenic contractions in a separated colon are much less frequent and complete than are those of the ileum, and peristaltic waves commencing in the first part of the large bowel seldom reach so far as the sigmoid flexure.

The precise nervous control of the gastro-intestinal movements is not accurately known. It would seem as if the governance of the automatic acts depended on the intrinsic ganglia (Auerbach's and Meissner's), and as a rule the vagus influence is that of an excitant and the sympathetic that of inhibition; but this is certainly not invariably so with the pyloric sphincter. In the colon the fibres from the spinal nerves correspond to the vagus above. The bile appears to be a normal stimulus of intestinal peristalsis and even of the contractions of the plain fibre cells in the villi, whereby the terminal lacteal radicles are compressed and the contents moved.

When the contents of the colon from which most of the fluid has been absorbed reach the lower end of the bowel, their further progress is very much impeded by the curves of the sigmoid and the "valves" of the rectum, and they lodge in that situation. When *defæcation* takes place a deep inspiration is taken, and the abdominal muscles forcibly contract against a full chest and closed glottis. This forces the fæces from the upper into the lower part of the rectum, where their presence

reflexly excites strong peristaltic movements, which extrude the material through the relaxed sphincters, the anal canal coming more into line with the rectum. The tonicity of the sphincters, which may be increased or diminished by impulses from the cerebrum (emotion, etc.), or reflexly, depends upon the lumbar enlargement of the cord being intact.[1]

Absorption.—It is all-important for the continued and effective action of the digestive secretions that the digesta should be removed without delay. As a rule very little absorption takes place by the gastric mucous membrane, although some salts, sugars, peptones, and alcohol do enter the blood from the stomach, especially the last named. In this restricted absorptive capacity of the organ may be seen a protection to the body generally, supplementing the further protective power exercised by the germicidal action of its secretion. The absorptive power of the stomach is, however, of some value, as indicated by the experiments of Heidenhain, which go to show that the efficiency of the gastric secretion is partly dependent on materials absorbed at the commencement of digestion. It is, however, from the small intestine that the greater part of the absorption takes place, chiefly by the villi, the capillaries of which take up the proteids, carbohydrates, salts, and water, the fats finding their way into the lacteals. But, although no part of the subject of digestion proper, it is to be noted that most of these alimentary principles experience further change in their passage through the columnar epithelium. There the albumoses and peptones are reconverted into coagulable proteids, in which form they are found in the blood; a small portion also of the nitrogenous food which has escaped digestion is also absorbed as such. The carbohydrates which are converted into maltose and dextrin by digestion are in the form of dextrose in the blood of the portal vein and in the chyle, having probably undergone conversion in their passage through the epithelium, or in the tissues of the villi. Still more distinct are the changes in the fats, most of which, becoming soaps in the intestine, are found in the lacteals as neutral fats similar to those originally ingested. Some effect upon many of the salts appears to take place in the course of their transference from bowel to blood current. The water is mainly absorbed from the lower part of the canal, especially from the colon, the intake being much increased by the secretions poured out.

The fæces.—By the absorption of the fluid portion the contents of the colon become increasingly consistent in their progress along the bowel, and are evacuated as a solid cylindrical mass averaging daily about five ounces in weight, though the quantity varies much with the character of the food, of which the fæces form one-seventh to one-eighth by weight. The normal colour is due to a derivative of bilirubin, which is the chief pigment of the bile, known as stercobilin (urobilin); but it varies much within the limits of health, being largely influenced

[1] See footnote, p. 218.

by the food, being much darker on a purely meat diet, or if the fæces be long retained, and of a brighter yellow with milk foods, and often greenish if much vegetable be taken. It is important to note that unaltered bile pigment is not found normally in the stools. In reaction the motions are slightly acid or slightly alkaline, also dependent on the nature of the ingesta. The fæces consist of indigestible food residue, such as seeds, bones, skins of fruit, etc.; portions of food which have escaped digestion; unabsorbed products of digestion, *e.g.* leucin, tyrosin, indol, skatol, to which the fæcal odour is due; excretory substances chiefly derived from the bile, such as cholesterin and stercobilin; earthy salts; mucin and the desquamated epithelium and detritus from the surface of the mucous membrane, with micro-organisms.

For the efficient performance of these secretory, motor, and absorbent processes it is obvious that a satisfactory state of general nutrition, an unimpeded and adequate blood-supply, and even more especially a healthy and undisturbed nervous control, are essential. Exertion, whether mental or bodily, directly after a meal, is apt to be harmful, probably by interfering with the blood circulation. The same may be said for sleep under like circumstances, although sleep appears to exert no influence on either gastric or pancreatic secretion (Pawlow).

It is also noteworthy that in the healthy state the activities of the stomach and intestines are carried on without conscious sensation to the individual. The healthy appetite or desire for food is characterised by peculiar sensations referred to the epigastrium, together with a general "sinking feeling," and appears in great measure to be connected with the condition of the stomach. It is commonly allayed by the taking of sufficient food; but it is not a mere question of the stomach being empty or full, since there may be anorexia when the viscus is quite empty, but the mucosa is unhealthy, or the desire for food may quickly follow a full meal. Even more than with many other of the bodily functions, the influence of habit is potent in modifying the processes of digestion within limits that are not detrimental to the individual's well-being. Moreover, it seems probable that by the long continuance of a certain diet a permanent form of gastric or pancreatic activity might be established, and that abrupt changes of food might thus lead to digestive failures.

Lastly, there is a function of the stomach, and possibly of the intestines, of which but little is positively known, and still less what share it may take, if any, in normal digestion, and that is its excretory power. Various substances, for the most part toxic, such as antimony, morphia, toxalbumins, and toxalbumoses, have been found to pass from the blood-vessels of the stomach into the organ, and then to produce their poisonous effects by local irritation or by reabsorption.

W. H. Allchin.

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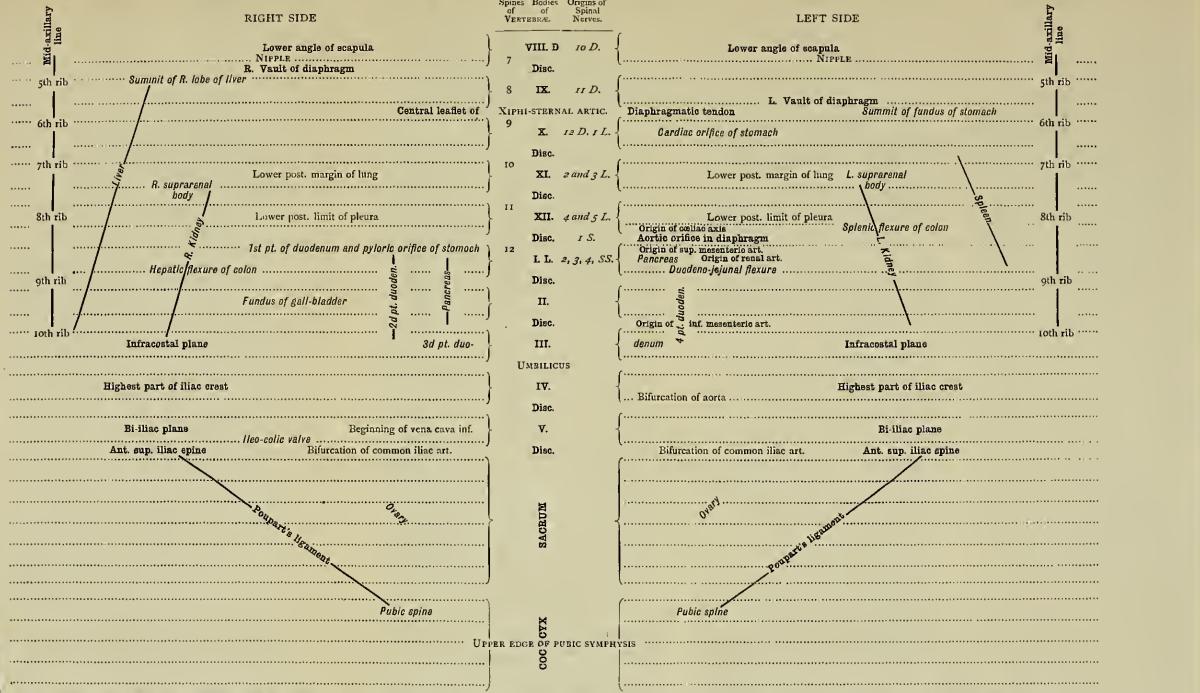
TABLE SHOWING THE LEVELS OF VARIOUS STRUCTURES IN THE ABDOMEN IN RELATION TO THE SPINES AND BODIES OF THE VERTEBRÆ



Adapted by the Editor from a similar table in Quain's *Anatomy* with the assistance of Prof. Thane; many of the positions are to be regarded as only approximate by reason of individual variations.

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FOOD AND DIET

The use of food is to provide material (1) for the repair of tissue waste; (2) for the production of work and heat.

Chemical analysis shows that every true food contains one or more groups of compounds, in virtue of which it is able to fulfil the above functions. These compounds are termed the *nutritive constituents* of the food, and may be divided into the following groups:—(1) water; (2) mineral matters; (3) proteids (*e.g.* egg-albumin, myosin, etc.); (4) albuminoids (*e.g.* gelatine); (5) fats; (6) carbohydrates (*e.g.* sugar and starch).

The first function of food, that of repairing tissue waste, can only be performed by the first three of these constituents—water, mineral matter, and proteids. These may, therefore, be termed "tissue builders." The second function, that of producing energy (heat and work) can be fulfilled by the proteids, albuminoids, carbohydrates, and fats, though in different measure. These may, therefore, be termed "energy producers."

It is evident that proteids alone can fulfil both the functions of a food, and of the proteid which we eat part is devoted to the repair of tissue waste, part is consumed in the production of heat and work. The larger the amount of albuminoids, carbohydrates, and fats supplied along with the proteid, the smaller will be the fraction of the latter which is converted into heat and work, and the greater the share which can be devoted to making good wear and tear. The former groups are, therefore, spoken of as "proteid sparers," and the greater the amount in which they are represented in the diet the less proteid do we require. This explains the possibility of living upon a diet consisting almost solely of potatoes, which though containing very little proteid, are peculiarly rich in carbohydrates. Of the three groups mentioned above the albuminoids are the most efficient sparers of proteid, then the carbohydrates, and lastly the fats. It will be evident, however, that as long as life lasts a certain amount of proteid must be supplied, for that constituent alone is capable of replacing the waste of nitrogenous matter in the body which is constantly going on. Hence the paramount importance of providing for a due supply of proteid in the body both in health and disease.

In judging of the *value of a food* we have to consider (1) the proportion of nutritive constituents which it contains, *i.e.* its chemical composition; (2) the amount of energy (heat and work) which it is capable of yielding; (3) its ease of digestion and absorption in the body; (4) its cost.

Chemical Composition of Foods

The following tables show the percentage composition of the chief articles of food in common use :[1]—

Composition of Animal Foods

Food.	Water.	Proteids and Albuminoids.	Fat.	Mineral Matter.
Beef (moderately fat) . .	70.8	22.5	4.5	1.2
Beef (roasted)	55.3	34.2	8.2	1.4
Mutton (moderately fat) .	75.0	18.0	5.7	1.3
Pork (moderately fat) . .	60.9	12.3	26.2	0.6
Veal	71.0	17.0	11.0	1.0
Bacon	22.3	8.1	65.2	4.4
Fowl	70.0	23.3	3.2	1.0
Kidney	78.7	16.8	3.2	1.3
Liver *	61.2	23.1	9.0	1.7
Sweet bread	70.9	16.8	12.1	1.6
Blood	80.8	18.1	0.2	0.8
Tripe	74.6	16.4	8.5	0.5
White Fish	80.4	14.0	2.0	3.6
Salmon (raw)	71.5	18.7	6.2	0.6
Salmon (boiled)	63.3	25.9	5.9	0.8
Oysters †	86.3	6.1	1.4	1.9

* Liver contains also about 5% of carbohydrate in the form of glycogen.
† Oysters contain about 3% of glycogen.

Milk and its Derivatives : Eggs

	Cow's Milk.	Butter Milk.	Koumiss.	Egg (white).	Egg (yolk).	Egg (whole).
Water . . .	87–88%	92.9	90.7	85.7	50.9	73.7
Proteid . . .	2–3	2.3	2.2	12.6	16.2	14.8
Fat	$3\frac{1}{2}$–$4\frac{1}{2}$[1]	0.4	2.1	0.25	31.75	10.5
Carbohydrate .	4–5	3.7	1.5	...	...	...
Mineral matter	0.7	0.7	0.9	0.59	1.09	1.0
	Ordinary cream has 41% fat; Devonshire cream has 58%; butter has 82%.		Koumiss also contains 1.7% alcohol and 0.9% of lactic acid.			

[1] In those instances in the tables in which the percentages do not amount in the aggregate to 100 it is to be understood that the balance is made up of "extractives" and other non-nutritive ingredients.

Cheese may be regarded as consisting of about one-third water, one-third fat, and one-third proteid. It has usually 4% of ash, and contains also a small amount of milk sugar.

Cereals and their Derivatives

	Water.	Proteid.	Fat.	Starch and Sugar.	Cellulose.	Mineral Matter.
Whole Wheat Meal	12.1	12.9	1.9	70.3	1.6	1.2
Fine Flour . . .	13.0	9.5	0.8	75.3	0.7	0.7
White Bread . .	40.0	6.5	1.0	51.2	0.3	1.0
Whole Meal Bread	45.0	6.3	1.2	44.8	1.5	1.2
Fine Biscuits . .	1.18	13.31	3.18	81.08	0.25	1.0
Oatmeal	7.2	14.2	7.3	65.9	3.5	1.9
Pearl Barley . .	12.7	7.4	1.2	76.7	0.8	1.2
Maize Meal . . .	11.4	8.5	4.6	72.8	1.4	1.3
Rice	12.4	6.9	0.4	79.4	0.4	0.5
Italian Pastes . .	10.40	12.51	0.80	75.23	0.30	0.76
Semolina	10.50	11.96	0.60	75.79	0.50	0.65
Tapioca	12.80	...	0.20	86.88	0.08	0.04

Pulses

	Water.	Proteid.	Fat.	Starch and Sugar.	Cellulose.	Mineral Matter.
Green Peas	78.1	4.0	0.5	16.0	0.5	0.9
Dried ,, ...	13.0	21.0	1.8	55.4	6.0	2.6
Lentils	11.7	23.2	2.0	58.4	2.0	2.7
Haricots verts. . . .	89.5	1.5	0.4	58.6	0.6	0.7
,, blancs . . .	11.7	23.0	2.3	55.8	4.0	3.2
Soy Beans	11.0	32.9	18.1	28.7	4.4	4.9
Revalenta Arabica . .	9.1	22.0	1.5	65.2	...	2.2

Roots and Tubers

	Water.	Proteid.	Fat.	Starch and Sugar.	Cellulose.	Mineral Matter.
Potato	76.7	1.2	0.6	19.1	0.6	0.9
Turnip	90.3	0.9	0.1	5.0	1.8	0.8
Carrot	86.5	0.5	0.3	10.1	1.5	0.9

Green Vegetables, Fruits, etc.

	Green Vegetables.	Fresh Fruits.	Dried Fruits.	Nuts.	Mushrooms.	Irish Moss.	Iceland Moss.
Water	90.0	85 to 90	20	4 to 5	93.7	18.8	10
Nitrogenous Matter	1.0	0.5	2 to 5	15 to 20	3.5	9.4	8.7
Fat	0.4	0.5	2 to 4	50 to 60	0.3	...	...
Carbohydrates . .	3 to 5	5½ to 10½	65 to 75	9 to 12	1.2	55.4	76.3
Cellulose	1 to 2	2½	5	3 to 5	1.1	2.2	3.5
Mineral Matter . .	0.5	0.5	1 to 2	1	0.3	14.2	1.5

It will be observed that the animal foods, as a group, are characterised by their richness in proteid and fat; the vegetable foods by an abundance of carbohydrates and water. The vegetable substances which contain most proteid are the pulses and some of the cereals, such as oatmeal. The difference in nutritive value between the fat and lean fishes should be noted. Green vegetables, fresh fruits, and the lichens and fungi, are of low value, but the vegetable salts of potash which the two former groups contain, give them a special and important place in the diet.

Care must be taken not to be led astray by the use of the vague term "nitrogenous matter," often used in describing the chemical constituents of foods. "Nitrogenous matter" covers proteids, albuminoids, "extractives," and other bodies, and it makes all the difference which of these is referred to. Proteids are the only real "flesh-formers," albuminoids are of value as proteid sparers and a source of energy, while the "extractives" (which include such substances as creatin, xanthin, amido bodies, etc.) are of no real nutritive value at all. It should be pointed out that the composition of foods is considerably influenced by cooking. Animal foods become less, vegetable foods more watery after being cooked. Thus raw beef contains about 70 per cent of water, while the same beef after boiling has only 57 per cent, or when grilled 55 per cent, the solid constituents (proteids and fats) being relatively increased in proportion. It is for this reason, amongst others, that meat which has been very thoroughly cooked is less digestible than that which is underdone.

Amount of Energy yielded by Foods

The measure of the amount of energy which a food is capable of yielding is the Calorie, that being the amount of heat which is required to raise the temperature of 1 kg. of water 1° cent. The number of

Calories yielded by the combustion of one gramme [1] of each of the chief nutritive constituents of food is as follows :—

Proteid	4.1 Calories
Fat	9.3 ,,
Carbohydrate	4.1 ,,

In other words, a given quantity of fat will yield in the body fully twice as much energy as an equal amount of proteid or carbohydrate. This might also be expressed by saying that one part of fat is the *dynamic equivalent* of two and a quarter parts of starch, sugar, or proteid. Now as far as the cells of the body are concerned, it does not seem to matter much whether the energy they require is supplied as proteid, fat, or carbohydrate, provided always that the total number of Calories is sufficient. Theoretically, for instance, a diabetic ought to be able to get on quite comfortably if one replaces his carbohydrates by their dynamic equivalent of proteid or fat. In mild cases, indeed, this can be done, but in the severer forms of the disease one has to encounter the practical difficulty that the provision of a sufficiency of fat is very apt to overtask the patient's powers of digestion.

In order to ascertain the energy-yielding value of any diet all one has to do is to ascertain, by means of such analytical tables as those given above, the number of grammes of proteid, carbohydrate, and fat which the diet contains. By multiplying the figures obtained by the number of Calories yielded by one gramme in the case of each constituent one arrives at an estimate of the total amount of potential energy which the diet yields. This method is one which is now in constant use in gauging the value of dietaries in hospitals and other public institutions. If one compares, for instance, by this means the amount of energy which one would derive from the consumption of one pound of each of some typical foods, one would get something like the following result :—

Comparative Energy-Value of Foods

Food.	Calories as Proteid.	Calories as Fat.	Calories as Carbohydrate.	Total Calorie-Value per lb.
Butter . . .	18.0	3,559.0	none	3,577.0
Peas . . .	418.8	71.7	982.5	1,473.0
Cheese . .	553.0	750.0	none	1,303.0
Bread . . .	130.0	21.5	976.5	1,128.0
Eggs . . .	232.0	507.0	none	739.0
Beef . . .	391.0	232.0	none	623.0
Potatoes . .	18.5	9.3	341.2	369.0
Milk . . .	67.0	168.0	87.0	322.0
White Fish .	299.0	16.0	none	315.0
Apples . .	9.0	none	229.0	238.0

[1] One gramme = 15½ grains.

It would be obvious at a glance that the foods which are capable of yielding most energy are those which are richest in fat, and for this reason such articles must always occupy an important place in the diet, even in early life.

The Digestibility and Absorbability of Foods

By the *digestibility* of a food one means the ease with which it is reduced to a state of semi-solution in the stomach; the term "absorbability" is used to indicate the degree to which the constituents of the food are capable of being taken up into the blood.

The physical form of a food has more influence upon its digestibility in the stomach than any other consideration. Fineness of division greatly facilitates solution in the stomach. Hence the importance of good teeth to healthy digestion. On the other hand, all tough and hard articles are dissolved with difficulty, while the presence of much fat in the interstices of a food militates against rapid disintegration by preventing the ready ingress of gastric juice. It is for this reason that pastry and goose are articles which tax greatly the digestive powers.

Foods may be divided into the following four groups according to the ease with which they are digested in the stomach:—

1. Beef-tea, milk, soft or raw eggs, Albert biscuits, rusks, dry toast, oysters.
2. Boiled brains, sweet-breads, boiled fowl or pigeon, calves' feet.
3. Scraped underdone beef or mutton, potato purée, stale bread.
4. Roast chicken or game, roast veal, cold roast beef (underdone). white fish, macaroni, rice, chopped spinach.

The articles in the first group leave the stomach in from one to two hours; those in the last in from four to five hours. The quantity of any article eaten has less influence on its digestibility than its consistence. Doubling the quantity, for instance, does not mean doubling the time required for digestion, but only prolonging it by about one-half. Water leaves the stomach almost immediately it has been swallowed. The stay of other fluids is conditioned by the amount of solid matter which they hold in solution. It is important to remember that even a heavy meal, such as dinner, should have been entirely passed on out of the stomach after the lapse of five hours. If that fails to happen one is justified in assuming that the motive power of the stomach wall is defective. Certain of the accompaniments of meals have a distinct influence upon the rate of digestion. Water, however, is almost devoid of any influence unless the amount taken is excessive; hot water even accelerates digestion, and the same is true of most aerated beverages. Tea and coffee delay digestion both in the mouth and stomach, but alcoholic drinks have astonishingly little effect and, as far as the stomach, at least, is concerned, may even accelerate the process by promoting a flow of gastric

juice. Soup and all other articles which contain the extractives of meat and gelatin act in a similar fashion. For this reason it is right and natural to begin dinner with some warm soup, or even, in cases of fatigue, with a glass of sherry and bitters, and the use of beef-tea can be defended on this ground, if none other, that it promotes appetite and the power of digesting other food.

The *absorbability* of a food depends very much upon its chemical ingredients. The carbohydrates (with the important exception of cellulose) enter the blood most completely, and then the fats, whilst the loss of proteid is always somewhat greater. A mixed diet is usually better absorbed than any one article of food taken alone, and a purely vegetable regimen, owing mainly to the quantity of cellulose which it contains, leaves the greatest residue in the bowel, and by enclosing the nutritive constituents, excludes them from the solvent action of the digestive juices. This interference of cellulose with absorption is a point never to be lost sight of in estimating the value of vegetable foods. It explains the apparent paradox that whole-meal bread, though superior in a chemical point of view to white bread, is practically not such a valuable food as the latter. For cellulose is almost entirely incapable of digestion by man, the only important change which it undergoes being the conversion of part of it into marsh gas by bacterial action with the consequent production of flatus, a fact which explains the injurious influence of green vegetables and other foods rich in cellulose, in all cases in which there is a tendency to flatulence.

The following table gives a rough idea of the extent to which the nutritive constituents of different foods are absorbed :—

	Proteids.	Fats.	Carbohydrates.
Animal Foods . .	98%	97%	100%
Cereals and Sugars .	85%	90%	98%
Vegetables and Fruits .	80%	90%	95%

These facts have important bearings on the dietary suitable for intestinal disorders. In cases of diarrhœa it is advisable to give a food which will leave a small residue in the bowel. One should, therefore, select almost purely carbohydrate-containing foods, such as decoctions of barley and arrowroot. Underdone meat is also absorbed almost in its entirety. In cases of constipation, on the other hand, one should select articles which will leave a considerable residue to act as a stimulus to peristalsis. Here green vegetables and fruits find an appropriate place. It only remains to add that absorption is apparently affected to a comparatively slight degree by diarrhœa, and that such substances as the extractives of meat and vegetable acids have a stimulating influence on peristalsis, which contra-indicates their use in all such cases.

The COST OF A FOOD is an important practical question to be taken into account when dealing with its relative value, but can only be lightly touched upon here. It may be sufficient to point out that the market price of a food is regulated far more by aesthetic and other considerations than by its true nutritive worth. The cheapest food must necessarily be that which yields the greatest amount of building material (proteid) and energy for a given sum. Of the different nutritive constituents proteids and fats are the most costly. Hence, a diabetic diet must always be dear. The cheapest sources of proteid are skim milk, cheese, the coarser sorts of fish and, amongst vegetable foods, the pulses. The cheapest fats are margarine and dripping. These facts are of importance in constructing cheap meals. Of the two great classes of foodstuffs, animal and vegetable, the latter is in most cases by far the cheapest, a point which is one of the main arguments for vegetarianism.

THE AMOUNT OF FOOD REQUIRED IN HEALTH AND DISEASE

An examination of the excreta shows that about 20 grammes of nitrogen and 320 of carbon are required to replace the daily loss of these elements. Assuming that the whole of the nitrogen is derived from proteid, it would require about 125 grammes of the latter to make good the loss per day. This would also contain about 62 grammes of the necessary carbon; the remaining 240 grammes must be obtained from carbohydrate and fat. The exact proportions of these two constituents in the diet is to a large extent a matter of indifference, some individuals partaking more largely of fat, others of carbohydrate, depending upon taste, opportunity, and considerations of cost. As a rule, however, we may say that about 60 grammes of fat and 500 of carbohydrate are the correct proportions.

It will be obvious that no one article of food is likely to contain these ingredients in proper relative amount. Hence the necessity for a mixed diet. In addition to its purely nutritive constituents any satisfactory diet ought, of course, to contain a certain amount of flavouring ingredients, without which it would pall on the taste, and the proper secretion of the digestive juices would fail to occur.

The value of such a diet as the above in potential energy should be from 3000 to 3500 calories. This, of course, is the amount required by a man of ordinary weight doing a moderate amount of muscular work. Women require about one-fifth less food than men. The amount required by children has been estimated as follows (Atwater):—

A child under 2	requires	0.3	the food of a man	doing	moderate work.
„ of 3 to 5	„	0.4	„	„	„
„ 6 to 9	„	0.5	„	„	„
„ 10 to 13	„	0.6	„	„	„
A girl of 14 to 16	„	0.7	„	„	„
A boy of 14 to 16	„	0.8	„	„	„

The relatively large amount of food required by a child is necessitated (1) by the fact that growth is actively going on; (2) by the large amount of heat which a child loses owing to its relatively extensive body surface.

In old age the requirements in food are comparatively small owing to the inactivity of the cells at that period of life. Under-feeding is therefore the chief danger in childhood; over-feeding in old age.

It has not yet been definitely determined whether or not some individuals require, for the same output of heat and work, a larger supply of food than others. It is possible, however, that a few cases of obesity are to be explained by an economy of energy in the body.

Climate and season have comparatively little influence on the amount of food required. Any alterations in the heat-production or loss of the body which they may necessitate should be met by an increase or diminution of clothing, rather than by varying the quantity of food consumed.

Work and rest have, as might be expected, a more potent influence on the amount of food required than all other factors put together. This is true, however, of muscular work alone. Brain work does not in any way increase bodily waste. The demands of muscular work are best met by an increase of all the nutritive constituents, for even although proteid may not be required for the actual production of work —a point which is still *sub judice*—an increased supply of it is necessary to make good the greater wear and tear of tissue which severe labour implies. The importance of carbohydrates, and especially of sugar, as sources of muscular energy has recently received much attention from physiologists, and that constituent should certainly always find a large place in the diet of toil. The condition of a sick person, who is at rest in bed and whose loss of heat is greatly diminished, is such as to enable him to live upon much less food than one in health. An ordinary hospital diet for a full-grown man under these conditions need not comprise more than 2500 calories, a demand which would be met by the following scheme :—

Bread	10 ounces.
Potatoes	8 ,,
Meat (cooked)	6 ,,
Milk	1 pint.
Sugar	1 ounce.
Butter	1 ,,
Pudding	(rice 1 oz., 1 egg, sugar $\frac{1}{2}$ oz.)
Green Vegetables	6 ounces.

In treating cases of obesity it may be necessary to reduce the value of the diet to 1500 calories, or even less, the balance of energy required by the patient being obtained by the consumption of his own fat.

The effects of habitual *over-feeding* depend to some extent upon the particular ingredient of which an excess is taken. If this be proteid there is apt to be an accumulation in the body of the products of nitrogenous waste which tends to the production of gout and high arterial tension, while their elimination throws increased work upon the kidneys, and predisposes to chronic degenerative changes in these organs. Hence the diet of gout, high arterial tension, and renal disease should be one into which proteids enter rather sparingly. There is reason to suppose that the products of the disintegration of casein are less apt to produce these results than those of the nitrogenous matter of meat, and accordingly it is well for such patients to make a rather large use of milk and its derivatives. An excess of carbohydrates and fat in the diet is one of the main causes of obesity, and, in the case of the former ingredient at any rate, of those forms of chronic glycosuria often met with in elderly persons.

The general results of *under-feeding* are, on the whole, more serious than those just mentioned, and are particularly apt to be displayed in early life. A deficiency of proteid is more injurious than that of the other ingredients, and tends to lead to impairment of growth and to poverty of blood and muscle. A scarcity of fat in the diet tends in early life to the production of rickets, and later on there is little doubt that it predisposes to the attacks of the tubercle bacillus. Deficiency of carbohydrate alone is so uncommon that one can hardly assign to it any constant pathological effects.

Mineral Constituents of the Diet

The mineral ingredients of the food are to be regarded as important tissue builders. They are only very indirectly, if at all, sources of energy. Death ensues within almost a month if no mineral constituents are supplied, but it is impossible to state exactly how much of them is required daily. While there is no doubt that many mineral substances can be absorbed in an ordinary inorganic form it is certain that the organic combinations of mineral ingredients which exist in ordinary foods are of peculiar value in nutrition. The following are the chief mineral substances required by the body, with a list of foods richest in each :—

Calcium, in milk, eggs, cereals (especially rice), and some vegetables, *e.g.* radishes, asparagus, and spinach. *Iron*, in yolk of egg, red meats, Egyptian lentils, and oatmeal. *Phosphorus*, abundant in all animal foods, but especially in yolk of egg, sweet-breads, and fish roe. Also in the germ of wheat (*e.g.* Hovis bread). *Sulphur* is contained in all proteids. *Iodine* is present in minute quantity in fish. *Fluorine and silica* are derived from the cereals. Salts of *potash* are useful not merely in helping to build up muscles and blood, but also, when

combined with vegetable acids, in helping to maintain the proper degree of alkalinity of the blood. It is for the latter reason that fresh vegetables and fruits, which are such abundant sources of such salts, are an essential constituent of any properly constructed dietary. *Sodium* and *chlorine* are chiefly obtained in the form of common salt. While an ordinary mixed diet certainly contains sufficient salt for all the needs of the body, there is no reason to suppose that its moderate addition to the food in the form of a condiment is in any way injurious to health. *Calcium oxalate* is peculiarly abundant in such vegetable foods as tea, coffee, spinach, rhubarb, and sorrel, and it may therefore be sometimes necessary to forbid the use of such articles in cases of gravel.

Whilst, as stated above, we have no certain information as to the exact amount of the different mineral ingredients required daily, there is no doubt that a mixed diet, which is satisfactory in other respects, will contain all the inorganic matter which the body requires. The addition to the food of artificial mineral compounds such as phosphates, etc., is therefore uncalled for and unnecessary.

Beverages

Water.—About $2\frac{1}{3}$ pints of water are required in the liquid form daily. Restriction of the amount of water in the diet has, in conditions of health, very little effect on the composition of the blood, for the balance is quickly made up by absorption from the tissues. In dropsical conditions, however, a dry diet may aid in ridding the tissues of their excess of water, and so play a part in the therapeutics of cardiac disease. In cases where the heart's action is embarrassed, too, the sudden entrance of any considerable quantity of fluid into the circulation increases the work of the heart, and in these circumstances it may be well to see that only small quantities of water are taken at one time. The restriction of the amount of fluid consumed is also advocated by many writers as an aid in the treatment of obesity, but probably only really necessary when the condition is complicated by feeble action of the heart.

A good drinking water should be devoid of colour and odour, and have a pleasant fresh taste. It should contain only a moderate amount of solid matter, $8\frac{1}{2}$ grains per gallon being a good average. There is no satisfactory proof that the drinking of hard water is in any way injurious to health, equally little that soft waters tend to produce softness of bone. It is well to remember, however, that soft waters are peculiarly liable to metallic contamination. The only certain method of rendering safe a water which is bacteriologically suspicious is by boiling, with subsequent aeration in a gazogene. No form of filter can be relied upon with confidence to effect this result.

Water has little or no influence either upon digestion or absorption, and is itself not absorbed in the stomach. For this reason patients with pyloric stenosis are very apt to suffer from a deficiency of water in the blood.

Mineral Waters.—These may be divided into two classes—(1) artificial, (2) natural.

1. The artificial group consists either of (*a*) plain or aerated water charged with carbonic acid gas; (*b*) water holding in solution certain chemical salts, *e.g.* bicarbonate of soda or potash (5-15 grs. per pint), lithia water (5 grs. of carbonate of lithia per pint), etc., or combinations of various salts in imitation of different natural waters; (*c*) sweetened or flavoured mineral waters, *e.g.* lemonade, etc. Most of these contain about an ounce of sugar to the pint, with the addition of some acid flavouring substance; generally a mineral acid is used.

2. The natural mineral waters are a large and important group, most of which contain some sodium chloride along with carbonates of soda and lime. The carbonic acid gas which they contain is better dissolved than in the artificial waters, and accordingly they become flat less rapidly. Mineral waters, with the exception, perhaps, of the sweetened group, have a favourable influence on digestion. They should be avoided, however, in cases where there is a tendency to flatulent distension of the stomach, in cyanosis, and when the heart is embarrassed. The sweetened varieties may prove injurious in dyspepsia, especially in the gouty forms.

Tea, Coffee, and Cocoa.—These may be termed *alkaloidal beverages* from the fact that they contain either caffeine or theobromine. A tea-cupful of ordinary tea infused for five minutes contains about 15 grains of solid matter, including about 1 grain of caffeine and 2 or 3 grains of tannic acid. China tea yields less tannic acid than any other, and the proportion of the latter ingredient is also greatly influenced by the duration of infusion. A cupful of black coffee contains, as a rule, about the same amount of caffeine and tannic acid as an equal quantity of tea.

The composition of an average sample of pure soluble cocoa is about as follows:—

Moisture	4 per cent.
Nitrogenous matter	20 ,,
Fat	26 ,,
Other non-nitrogenous matters	46 ,,
Mineral matter	6 ,,

and has the following probable percentage of nutrients:—

Proteid	12 per cent.
Fat	26 ,,
Carbohydrates	25 ,,

Tea and coffee both interfere with salivary and peptic digestion by

the tannic acid which they contain. Hence a dyspeptic should select a China tea, and infuse it for as short a time as possible. Tea also tends to the production of a flatulent form of atonic dyspepsia, often met with in women, probably in part through its action on the nervous system. Both tea and coffee should be avoided as accompaniments to meals into which meat enters in any quantity. The oily matters which are produced in the roasting of coffee are also apt to prove harmful in cases of chronic gastric catarrh. Cocoa has less influence on digestion than either tea or coffee, though the amount of fat in it is apt to render it injurious to some people. Tea and coffee, owing to the caffein or volatile oil which they contain, are both nervous stimulants, and tend to remove the sense of fatigue and facilitate the action of the brain. They should be avoided in sleeplessness and only partaken of sparingly by "nervous" persons. Neither tea nor coffee has any power of lessening tissue waste. Indeed they tend rather to increase it. Even cocoa, though theoretically of considerable nutritive value, cannot be regarded as a real food, because so little of it can be taken at one time.

ALCOHOLIC BEVERAGES.—The physiological action of alcohol may be summarised as follows :—

1. Locally it acts as a chemical irritant. Hence the pharyngitis and gastritis of neat spirit drinkers.

2. Its action on the chemical part of digestion is only slightly retarding, while its influence on the mechanical processes is positively favourable. Hence the frequently valuable aid which alcohol affords in cases of feeble digestive power.

3. It acts as a temporary stimulant to the heart, increasing both the frequency and force of the beats. Hence its use in conditions of failing circulation.

4. It dilates the peripheral blood-vessels and causes the body to lose heat. This is the secret of much of its value in fever.

5. It is oxidised almost completely in the body and spares some fat in the process, but has probably no power of diminishing the consumption of proteid. For these reasons it should be avoided in obesity, but is of value in diabetes, and has no appreciable influence in restraining the destruction of the nitrogenous tissues which is such a marked feature in the metabolism of fever.

6. About one to one and a half fluid ounces of absolute alcohol is as much as can be completely oxidised by the tissues of most persons in one day, though personal peculiarity plays a considerable part in this matter. Such a quantity of alcohol would be contained in the following amounts of some of the commoner alcoholic drinks :—

Brandy or whisky . .	2	fluid ounces	(1 sherry-glass).
Port, sherry, and other fortified wines	5	,,	(2½ glasses).
Claret, hock, and other natural wines	10	,,	(one tumblerful).
Bottled beer . . .	20	,,	(one imperial pint).

7. Unoxidised alcohol is a tissue irritant, and for that reason alcoholic drinks should be forbidden in cases of renal disease, cirrhosis of the liver, and vascular degeneration.

8. The use of alcohol is unnecessary in health, but it may prove useful and an aid to digestion in conditions of fatigue and over-work.

Spirits act upon the body partly by their alcohol—of which they contain nearly 50 per cent.—partly in virtue of the bye-products of fermentation which they contain. These bye-products are of the nature of ethers, and seem to have a peculiar power of stimulating the nervous system. They are particularly abundant in genuine liqueur brandy, and accordingly in cases in which such an action on the nervous system is desired, *e.g.* in low forms of fever, that spirit should be selected. If the physiological effects of alcohol alone are desired, any pure spirit will do equally well.

Malt liquors, containing as they do from 1 to 2 ozs. of carbohydrate matter per pint, and from 3 to 5 per cent of alcohol, are the most truly nutritive of all alcoholic drinks. For this reason they are specially injurious in cases of obesity, but may be useful as an adjunct to meals, if one wishes to fatten a patient up. The derivatives of hops which they contain confer upon them also the power of exercising a sedative influence on the nervous system, and for that reason bottled stout is often of great service in cases of "nervousness" and insomnia. Being bulky drinks they should be avoided in all cases in which weakness of the heart exists, and they also tend to aggravate any chronic inflammation of the genito-urinary tract.

The most convenient classification of *wines* is into *natural* and *fortified*. The former, such as claret, hock, and Hungarian and Italian wines, contain not more than 12 per cent. by weight of alcohol; the latter, such as sherry, port, and Madeira, may have anything up to 20 per cent., or even more. The natural wines are alone suited for habitual dietetic use. The fortified wines, being richer in alcohol and also in ethers, are more valuable as stimulants in disease, though the larger proportion of sugar which they are apt to contain may render them harmful in cases of acid dyspepsia. To the gouty, on the other hand, it is probably the larger amount of alcohol in such wines rather than their sugar which renders them harmful.

Champagne is the best example of the *effervescing* wines. Strictly speaking, it is a purely natural product containing from 9 to 12 per cent of alcohol by weight, but as drunk in this country it is usually slightly "fortified." A *dry* brand contains only about 2 per cent of solid matter. Carbonic acid favours the absorption of alcohol, and for that reason champagne is a rapid stimulant, and as the effervescence exercises a sedative influence upon the stomach, it is specially indicated in cases in which there is a tendency to vomiting.

Cider and Perry may be regarded as natural wines peculiarly rich in

vegetable salts of potash. The oxidation of these salts in the body and their excretion by the urine in the form of carbonates may cause such beverages to be of use in cases of gravel, but their acidity is a cause of colic in many persons.

Medicated wines are concoctions of uncertain origin and composition, containing beef extract, malt, cocaine, and other substances.

Artificial and Proprietary Foods

1. *Beef extracts* are made by extracting lean meat with hot water and evaporating the solution to a paste. For practical purposes they may be regarded as consisting of water, mineral matter, and the extractives of meat. It is true that they may contain small quantities of soluble proteid as well, and in the case of some of them, such as Bovril, some of the fibre of the meat is also added, but the amount of nitrogenous matter of this nature which they contain is in no case sufficient to affect their nutritive value.

The only definite physiological action which can be assigned to beef extracts is that of increasing appetite and promoting a flow of gastric juice. They are in no sense foods, and though commonly regarded as stimulants, any effect of that sort which they may possess is probably to be attributed to the hot water in which they are usually dissolved. Their only use in feeding the sick is as flavourers and aids to appetite. For reasons already mentioned they should be avoided in cases where there is a tendency to diarrhœa and in renal disease, gout, and high arterial tension.

2. *Beef juices* differ from meat extracts in containing a proportion of soluble proteid. Usually this does not amount to more than 10 per cent of the whole juice, while the extractives and mineral matters are present in such amount as to preclude the administration of other than small quantities of the preparation. Hence it is that they are of no real value in aiding nutrition and, practically, are of no more use than the extracts. Egg-white, which contains 12 per cent. of proteid, if flavoured with a little ordinary extract, is vastly cheaper and quite as useful as any of them.

3. *Beef-tea* when made in the ordinary way does not contain more than 2 per cent of proteid, though if some of the fibre of the meat be grated down and added to the tea that proportion can be trebled or even quadrupled. Its nutritive value is superior to that of either of the above groups of preparations, simply for the reason that it can be swallowed in far larger quantity than they. Practically, however, it is of little real use as a *food*, though it is certainly a promoter of appetite and digestion. The contra-indications to its use are the same as in the case of beef extracts.

The following table shows the approximate composition of some typical examples of the above groups of artificial foods compared with fresh lean beef :—

	Water.	Proteid.	Gelatine.	Extractives.	Mineral Matter.
Lean Beef . . .	75.90	18.36	1.64	1.90	1.30
Liebig's Extract . .	18.35	9.45		29.67	23.68
Bovril	38.10	12.24		12.04	17.87
Natural Raw Beef Juice	91.10	5.10		3.10	...
Valentine's Beef Juice .	51.21	9.65		11.16	10.84
Brand's Beef Juice . .	59.15	15.45		16.55	8.85
Good Beef-tea . .	96.30	2.00		1.50	0.20

4. *Peptone preparations* contain proteid in a digested form, mostly as albumoses. There is no doubt that these are of as great nutritive value as ordinary proteid and as readily absorbed, but have the practical disadvantage of being apt, in large quantities, to produce diarrhœa. *Somatose* is one of the best of the solid peptone preparations ; it contains from 60 to 70 per cent of soluble proteid, and a teaspoonful of it may be reckoned as equal in nutritive value to about half an ounce of lean meat. Being practically devoid of taste it may be added to other foods without the knowledge of the patient.

Amongst fluid preparations of peptone are Denaeyer's (12 per cent); Brand's (7 per cent); and Darby's fluid meat (30 per cent). These contain in addition a considerable amount of extractives.

Peptonised milk is one of the most convenient forms in which to administer pre-digested nutriment. The following is the best way of preparing it :—

Dilute one pint of milk with a quarter of a pint of water. Heat to 140° F. Add two teaspoonfuls of *Liquor Pancreaticus* and twenty grains of bicarbonate of soda, mix, and place in a covered jug near the fire. At the end of one or one and a half hours bring to the boil, and keep till wanted.

A similar result can be obtained by keeping a pint of milk diluted with half a pint of lime water (to which three teaspoonfuls of *Liquor Pancreaticus* have been added) at the temperature of the room for three or four hours.

Peptonised milk gruel is made by mixing with thick oatmeal gruel, while still hot, an equal quantity of cold milk. To each pint of the mixture add two teaspoonfuls of *Liquor Pancreaticus* and twenty grains of bicarbonate of soda. Keep in a warm place for two or three hours, then boil and strain.

Of the peptone preparations as a class it may be said that they are only of use in increasing the proportion of proteid in the food temporarily, and when there is reason to suppose that the digestive powers are much impaired. Otherwise they are of no great assistance in feeding the sick.

5. *Casein preparations* are of recent introduction, and rank amongst the most useful of all artificial foods. They are prepared by isolating

pure casein from milk and rendering it soluble by incorporation with an alkali. They form colourless, tasteless, and odourless powders, easily soluble, and containing about three-fourths of their weight of pure proteid, the remainder being water and ash. Casein so prepared forms the basis of the foods manufactured by the Protene Company. Of other preparations similarly made "Plasmon" is the best and cheapest. As a means of artificially enriching the diet in proteid the casein preparations are unsurpassed, especially as their ready solubility and freedom from taste enables them to be easily added to other foods.

6. *Malt extracts* are artificial, and in a sense predigested, carbohydrate foods. An average sample has the following composition :—

Malt Sugar . . .	50 to 55 per cent.
Soluble Starch . . .	10 to 15 ,,
Proteids	5 to 6 ,,
Ash	1 to 2 ,,

They contain also the starch-splitting ferment, diastase.

Malt extracts may be prescribed with one of two objects : (1) to add to the fuel value of the diet. In this respect a dessert spoonful of such an extract as the above is about the equivalent of one egg, though vastly inferior to the latter, it must be remembered, in building material, while the fuel value of an equal quantity of honey is considerably greater ; (2) to aid in the digestion of starch. It may be questioned if this object is often present to the mind of the prescriber, and even did he have it in view it would be simpler and better to order a home-made infusion of malt, which could be drunk with meals that contain starchy food. *Honey* contains about 75 per cent of invert sugar along with traces of proteid. It is therefore actually richer than malt extract in carbohydrates, and contains these in a completely, instead of only a partially, pre-digested form.

7. Amongst *artificial fatty foods* are cod-liver oil emulsions, oil and malt, Lipanin, Virol, Pancreatic Emulsion, etc. The amount of fat in these rarely exceeds 50 per cent, and it is exceedingly doubtful whether they are in any way superior to cream or butter as foods.

Rectal Feeding

The best ingredients for *nutrient enemata* are (1) peptones and albumoses ; (2) raw beef juice ; (3) eggs with the addition of salt ; (4) dilute solutions of grape sugar ; (5) unboiled starch ; (6) alcohol.

Even under the most favourable conditions one cannot hope by this means to convey to a patient more than one-third of the amount of nutriment required even by a man at rest in bed. Hence rectal feeding is inevitably synonymous with slow starvation.

The basis of the enema may usually consist with advantage of peptonised milk, to which may be added peptone (1-2 ounces), grape sugar (1 ounce), or one or two eggs, to each of which a pinch or two of salt has been added, for that is found greatly to aid absorption. Red wine is a good form in which to give alcohol.

The whole bulk of the enema should not exceed eight ounces, and it should be given at the body temperature, through a small œsophageal tube passed as high up the bowel as possible, and fitted with a funnel. It should be administered very slowly. Three enemata (at most) in the twenty-four hours are sufficient. One cleansing injection should be given daily in addition.

Nutrient suppositories are not to be recommended. Their food value is almost *nil*, and their absorption uncertain.

Subcutaneous Feeding

Sterilised olive oil may be injected under the skin or into the muscles by means of an anti-toxin needle.

About 10 c.c. may be given at one spot, and three injections administered in one day. There is apparently no danger of producing fat embolism, while the oil is certainly utilised, and may in some cases be a useful accessory to rectal feeding.

Horse serum heated to a temperature of 55° C., at which point it is sterilised without being coagulated, has been suggested as a means of administering proteids subcutaneously. As much as 100 c.c. can be injected at a time without bad effects. The amount of proteid which can be given by such a method, however, amounting as it does to not more than 10 grammes, is too small to be of appreciable nutritive value, even assuming that the whole of it is utilised.

Artificial Feeding of Infants

The best substitute for human milk for infant feeding is cow's milk properly diluted. The milk of the ass, though sometimes of use temporarily, is too poor in fat to be suitable for any length of time. The following table shows the proportion of nutrients in cow's milk, human milk, and cow's milk diluted with half its volume of water :—

	Human Milk.	Cow's Milk.	Cow's Milk 2 parts. Water 1 part.
Proteid	1.5	2.5	1.6
Fat	3.5	4.0	2.6
Sugar	6.5	4.5	3.0
Mineral matter	0.2	0.7	0.4

If half an ounce of thin cream and two level teaspoonfuls of white or milk sugar are now added to every eight ounces of the mixture, the resulting fluid will be almost identical in quantitative composition with human milk. It is better to scald the cow's milk before use, as the risk of pathogenic infection is thereby diminished, while the chance of producing scurvy by such a proceeding is so slight as to be negligible. Barley water, or, better still, lime water, may be used for diluting instead of plain water. The mixture should be given at the body temperature, out of a boat-shaped bottle *without a tube*, in the amounts, and at the intervals shown in the following scheme :—

SCHEDULE FOR INFANT FEEDING

Age.	Intervals by Day.	Night Feeds.	Quantity at each Feed.
5th to 7th day	2 hourly	2	1–1½ ounces
2nd and 3rd weeks	,,	2	1½–3 ,,
4th and 5th weeks	,,	1	2½–3½ ,,
6th week to 3rd month	2½ hourly	1	3–4½ ,,
3rd to 5th month	3 ,,	1	4–5½ ,,
5th to 9th ,, ...	3 ,,	0	5½–7 ,,
9th to 12th ,, ...	3½ ,,	0	7½–9 ,,

In the case of very young or delicate infants it may be necessary to begin with a greater degree of dilution, *e.g.* equal parts of milk, water, and lime water, or two of milk to one each of water and lime water. If cream is unobtainable cod-liver oil emulsion may be given as a substitute, but it is better not to mix it with the milk.

In the case of infants who are unable to digest the casein of ordinary cow's milk even when freely diluted it may be necessary to use condensed milk for a time, as there can be no doubt that such milk is often more readily digested. One of the unskimmed milks should be selected, either unsweetened (*e.g.* Ideal, First Swiss or Viking), or sweetened (*e.g.* Nestlé's). From one to one and a half full teaspoonfuls of such a milk may be added to four ounces of water for each feed.

Fat and, in the case of the unsweetened milks, sugar must also be added. Though it must be admitted that such a proportion is theoretically too dilute, one finds as a matter of experience that a number of young infants do very well on it for a time at any rate.

Should this fail we must have recourse to peptonised milk, for the preparation of which Fairchild's peptogenic powder is useful. One may begin by peptonising for twenty minutes, and gradually reduce this time until the child is able to take the unaltered milk.

The numerous *patent infant foods* may be divided into the following groups :—

1. Desiccated milks with various additions, *e.g.* the Allenbury Foods, Horlick's Malted Milk, Nestlé's Milk Food, Carnrick's Soluble Food, etc.

2. Malted foods in which (*a*) the conversion of the starch has already been carried out, *e.g.* Mellin's ; or (*b*) is intended to take place during the preparation of the food for use, *e.g.* Benger's, Savory and Moore's, etc.

3. Unmalted farinaceous (starchy) foods, *e.g.* Ridge's, Neave's, etc.

The first of these classes is to be regarded as corresponding to condensed milk, and has no real advantage over the latter.

The second class may be of use as an addition to milk about the time of teething. Mellin's is the most suitable, unless great care is used in preparing the food for the infant.

The third class should never be used until the child has cut some teeth, and even then can only be regarded as expensive substitutes for such articles as rusks, stale bread, or oat flour.

R. HUTCHISON.

Fig 1

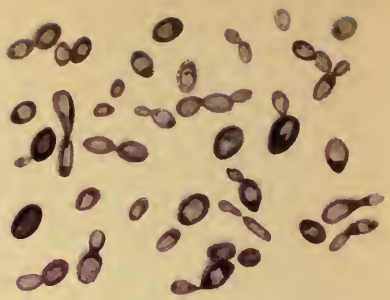

Fig 2

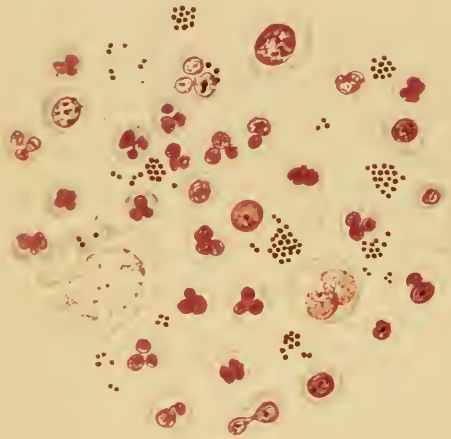

Fig 3

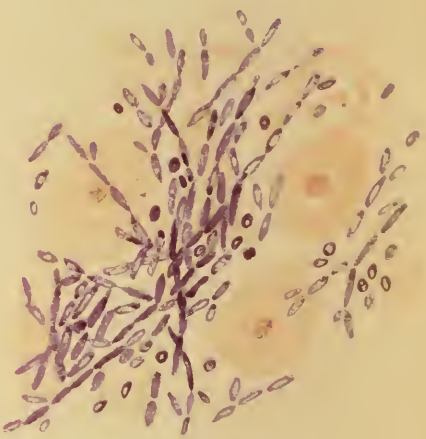

Fig 4

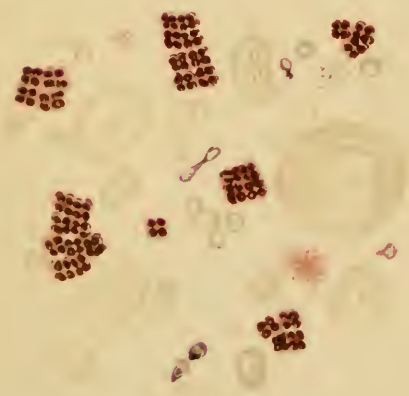

Fig 5

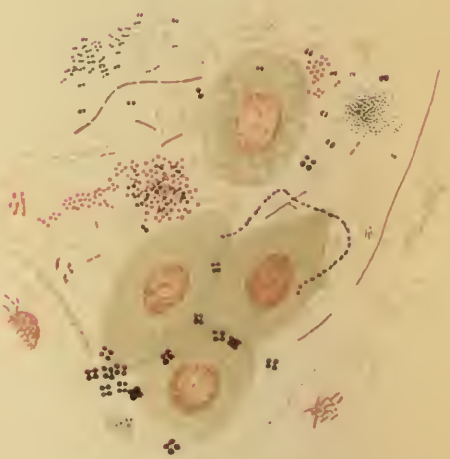

Fig 6

PLATE III.

FIG. 1.—Bacillus coli communis. Young agar culture, stained with fuchsin. × 1000 diams.

FIG. 2.—Cream-coloured Torula, cultivated from vomit. Stained with gentian violet. × 1000 diams.

FIG. 3.—Pus from periostitic abscess, containing Staphylococcus pyogenes aureus. Stained by Ziehl Neelsen's method, and with light green. × 1000 diams.

FIG. 4.—Oïdium albicans. Film from mouth of child suffering from "thrush." Stained with gentian violet and saffranin. × 400 diams.

FIG. 5.—Vomit from a dilated stomach, showing Sarcina ventriculi, Torula and Starch granules. Stained with hæmatin and picro-erythrosin. × 750 diams.

FIG. 6.—Fur, containing numerous micro-organisms, from tongue of child suffering from scarlet fever. Stained with eosin and methylene blue, Leishman's method. × 750 diams.

Drawn by Mr. R. Muir from Specimens kindly furnished to the Editor by Prof. Sims Woodhead.

THE BACTERIA OF THE ALIMENTARY TRACT

With the exception of the first few hours after birth the alimentary tract is always inhabited by bacteria. The length of time during which this aseptic condition persists is very short, for Escherich found bacteria in the meconium of infants twelve to eighteen hours after they had commenced to pass a separate existence, and never found that the stools were free from bacteria after the first twenty-four hours.

Whatever influence these organisms may have upon the functions of the alimentary tract and the parts in immediate relation with it, there is no doubt that life and the ordinary functions of life can be carried on in their complete absence. This has been conclusively proved by the experiment of Nuttall and Thierfelder, in which they delivered young guinea-pigs by an aseptic Cæsarean section, received them into sterile chambers, supplied them with sterile air and food, and proved the complete asepticity of the whole process by cultivation of the various excreta.

In the same direction point the experiments of Schottelius, who hatched and reared chicks aseptically. He found that their weight increased up to the twelfth day, and was then greater by 25 per cent than at birth. Nevertheless control chicks living under normal conditions throve far better. For whereas growth stopped in the aseptic chicks at the twelfth day, and was only one of 25 per cent, in the controls it was 140 per cent on the twelfth day, and on the seventeenth had reached to 250 per cent. Further, although the excreta of the experimental chicks were aseptic, there was present, after the fourth day, an enzyme that digested gelatine very slowly. Schottelius, therefore, considered it possible that the enzymes are incapable of exerting their full power until aided by bacteria, which appear normally in the excreta in thirty-six hours, for earlier than this no increase in body weight takes place.

The bacteria of the alimentary tract are of many kinds, and the varieties met with differ in different parts of the canal both in numbers and in species. Some of these micro-organisms are pathogenetic, the majority are non-pathogenetic, and some are pathogenetic for lower animals, but are innocuous to man. Some varieties are what may be termed normal inhabitants of the tract, while others—and amongst these are some of the most important—are intruders.

The main sources of the bacteria under consideration are the food and the respired air. Even in the case of the stomach and intestine, it must be remembered that swallowing of saliva, bronchial mucus, etc., to say nothing of definite sputum which has come from the lungs in such

diseases as phthisis and pneumonia, introduces to these parts bacteria that were originally aërial. The bacteria, however, which reach the alimentary canal by way of the mouth are not necessarily confined to the tract, for there is ample evidence that morbid conditions in other regions may sometimes be set up which are clearly traceable to invasion of the parts affected by organisms escaping from the alimentary tract. This is particularly noticeable in the case of the putrefaction of the body which sets in after death. That this change depends almost entirely upon escape of putrefactive bacteria from the intestines is shown by the fact that if the abdominal viscera (and especially the stomach and intestines) are removed from the body immediately after death putrefaction of the remaining parts is more or less postponed, and may be delayed to such an extent that the body passes into a condition of mummification without the occurrence of any appreciable putrefaction.

Whether the bacteria found in the alimentary canal are of service to the economy of their host, and, if so, how far this is the case, are questions to which no sure answers can as yet be given, though Schottelius presented some evidence in this direction in the experiments mentioned above. It is certain that in many instances they assist in the breaking down of proteid, but whether the less complex substances produced in this way can be and are utilised by the body at large for its nutrition is doubtful. The most that we know is that in life under completely aseptic conditions, as in the experiments already indicated, the fæces do not contain phenol, cresol, indol, skatol, or pyrocatechin, substances found in the fæces of animals living under ordinary conditions. On the other hand, aromatic oxy-acids are excreted in the urine, as they are under ordinary conditions, and must therefore be formed independently of bacteria. It is known, also, that in the test-tube, though certain varieties of bacteria break up proteid with the formation of albumoses, the formation of peptone itself is a far more uncommon result, if it occur at all. Complicated though the question is, the fact that infants who do not thrive with sterilised or humanised milk, sometimes begin to put on flesh directly their food is changed to dilute unsterilised cow's milk, may perhaps be considered as some indication that the relative absence of bacteria in the one case, and their presence in large numbers in the other, determines in great measure the readiness with which the proteids of the milk are broken up and the suitability for absorption of the resulting products. Upon the whole, however, the tendency at the present time is to believe that although the bacteria in the alimentary tract play an extremely important part in inducing intestinal putrefaction, they only act in this direction, and do not produce bodies which are utilised by the body to its advantage.

The mouth.—If a coverslip film be made and stained of saliva from a normal mouth, innumerable organisms of various kinds will be seen. Some are bacilli, either long filaments of thick bacilli or separate

delicate and thin rods, or short thick rods with rounded ends, and likely to show bipolar staining. Others are cocci, and may be staphylococci or streptococci, or, more rarely, diplococci. Others are spherical or oval, and much larger than micrococci; they stain deeply, and are usually present in somewhat scanty numbers (torulæ or yeasts). Occasionally there are seen branching filaments which are either streptothriceæ or higher mould fungi. On attempting to make cultivations from such normal saliva only a proportion of the above will grow. Nevertheless Sternberg gives a list of forty-eight non-pathogenetic and twenty-five pathogenetic varieties that have been isolated from the mouth, and Miller of Berlin has isolated and cultivated more than 100 different species. With the experience, however, derived from the examination of swabs taken from the fauces in cases of suspected diphtheria, one may say that the commonest bacteria of the mouth are staphylococci, particularly staphylococcus pyogenes albus and staphylococcus pyogenes aureus, that next to these come bacilli of various kinds, including the pseudo-diphtheritic bacillus, and that behind these come streptococcus forms, including streptococcus pyogenes, and various forms of torulæ.

It has been indicated that the greater number of those which have been isolated are non-pathogenetic, and even of those which are pathogenetic to animals, it is only a small number that is known to be the cause of disease in man. Apart from the pyogenetic cocci which are met with so frequently in the mouth that they may almost be considered as normal inhabitants, micrococcus pneumoniæ and B. diphtheriæ are not infrequently present in the mouths of persons who are apparently in perfect health. In the case of B. diphtheriæ this is particularly likely to be the case if the individual from whose mouth the culture is made has been in close relation with a person suffering from diphtheria (*e.g.* a hospital nurse). The same is true of B. tuberculosis, though to a far lower degree, for though tubercle bacilli are liable to be found in the saliva of nurses in institutions for consumptives, they are most decidedly not usual inhabitants of the mouth.

Apart from diphtheria and lobar pneumonia the presence of diphtheria bacilli and pneumococci in the normal mouth is important as bearing on the etiology of chronic rhinitis and otitis media. There is a certain amount of evidence to show that some forms of chronic rhinitis are associated with the presence of a bacillus indistinguishable from the diphtheria bacillus, and there is no doubt that a large majority of cases of otitis media depend upon an infection of the middle ear by pneumococci that have travelled thither from the mouth by way of the Eustachian tube. So far as the mouth itself is concerned, the evidence connecting definite diseases with definite varieties of micro-organisms met with in that region is very small except in the case of diphtheria. In the various forms of stomatitis, including the gangrenous form, streptococci

are met with in large numbers, it is true, or perhaps a diphtheria-like bacillus, but that the disease and the micro-organism in question are etiologically connected is a matter of the utmost difficulty to prove. Even in the case of the mild form of stomatitis which constitutes aphthæ of the mouth in ill-nourished infants, and which has long been known to be closely associated with the presence on the tongue of oïdium or saccharomyces albicans, it is not quite certain whether the micro-organism is to be regarded as the cause of the disease or only as accidental.

So far as concerns the propagation of the bacteria of the mouth, there is a certain amount of evidence that the saliva can act inimically to some varieties of bacteria. This evidence is partly positive, and consists of experiments made by Sanarelli, and partly indirect. For in fever, a condition in which one of the most definite changes is a diminution in the amount of saliva secreted, or perhaps a complete arrest of the flow (Dickinson), one of the chief constituents of the "fur" that accumulates on the tongue is an enormous thickness of bacteria of all kinds. Naturally the matter is not a simple one, but it seems as if, with the absence of the saliva, the bacteria of the mouth were able to take on an unrestrained growth; the nutrient material for the growth of the bacteria within the mouth being principally afforded by particles of food which remain after meals. This is a matter of the greatest importance, not only from the point of view of the ordinary putrefactive bacteria of the mouth which, by the products of their life history, have so considerable an influence upon the general health, but also from the point of view of those bacteria which are especially bound up with the questions of dental caries, acute and chronic alveolar abscesses and alveolar pyorrhœa.

The stomach.—It is not necessary to delay in order to consider the flora of the œsophagus, for, except in cases of malignant disease of the tube, bacteria are not present there in great numbers, and are identical in characters with those found in the mouth itself.

In the stomach the number and varieties of bacteria present vary considerably. Immediately after a meal they are much the same as those usually present in the mouth, with the addition of those which have been ingested with the food. But after they have been subjected to the action of the acid gastric juice for a little time, relatively few bacteria are found at all in the stomach, and of those which are present the greater number are dead. This is not the case, however, with some of the more resistant forms, such as B. tuberculosis and the spores of sporogenous micro-organisms, nor of those varieties, such as B. acidi lactici, and the bacillus of butyric acid fermentation, which normally grow to a certain extent in acid media.

An important point in connection with the question of the germicidal action of the gastric juice arises in the cases of B. typhosus and Vib.

choleræ asiaticæ. Kurlow and Wagner found that normal gastric juice destroys the vitality of the former species in one hour, and of the latter in half an hour outside the body. It is, therefore, clear that under exceptional circumstances (and particularly if the stomach be empty at the time of their ingestion) these micro-organisms might gain access to the stomach, and pass thence into the intestines of perfectly healthy individuals, and give rise to their specific diseases. Thus, a healthy laboratory boy on one occasion mistook a suspension of typhoid bacilli in water that had been prepared for class purposes for pure drinking water, and took a long draught. Shortly after he was laid up with a severe attack of typhoid fever. In this case the number of bacilli that were swallowed must have been enormously in excess of that which leads to typhoid fever contracted in the ordinary manner.

But though there is a great destruction of bacteria in the normal stomach, the case may be very different in diseases of various kinds. These diseases may be peculiar to the stomach, or they may affect primarily other parts of the body. In almost all of them, however, the fundamental factor in determining an alteration in the numbers of bacteria in the stomach is an alteration in the secretion of the free HCl of the gastric juice. For there is no doubt that the germicidal action of the gastric juice depends upon the free acid which it contains. In no inconsiderable number of diseases does the secretion of free HCl suffer such a diminution that the gastric contents are definitely alkaline for long periods, and in many others in which this is not the case the acidity is not occasioned by hydrochloric acid, but by lactic and other organic acids, the germicidal action of which is of the slightest. Under such conditions as these the bacteria reaching the stomach proliferate rapidly, and by their presence and their products tend to institute and perpetuate a vicious circle. In this way are to be explained many varieties of the disease summed up under the name "dyspepsia." A marked example of the statement just made is afforded by chronic dilatation of the stomach, in which the stomach contents are often distinctly alkaline, contain myriads of bacteria (especially sarcina ventriculi), and in which beneficial results often follow upon systematic washing out of the cavity. Nevertheless, it is doubtful whether any particular variety of micro-organism can be associated with any particular variety of dyspepsia at the present time. When much alkaline mucus is poured out into the stomach, as in the later stages of uncompensated heart disease, the number of bacteria in the stomach is usually very great. The same is true in cases of gastric carcinoma where there is a deficient formation of HCl.

Under special circumstances more or less specific forms of gastritis may be due to bacteria. Thus Dr. Cayley published a case of gangrenous gastritis due to streptococci; Mr. Foulerton, a case from which

pneumococci were isolated ; and examples of diphtherial gastritis, though rare, are not unknown.

The intestines.—The number of bacteria in the intestines varies greatly in different parts. Speaking broadly, it may be said that the number diminishes down to the ileo-cæcal valve, and increases from the ileo-cæcal valve to the rectum. This point has not long been known, and has been clearly brought out in a research upon the subject by Professor Lorrain Smith and Dr. Tennant of Belfast. Until recently the results of investigations have been obtained from the human cadaver ; but, as will appear later, there is the greatest reason for believing that results obtained after this method are not applicable to the subject in life and in health. The fundamental reason for the particular distribution of the bacteria in the intestines probably lies in the fact that, contrary to general teaching, the intestinal contents are acid throughout the small intestine, and only become alkaline when they have passed over some distance of the colon (Gamgee). These peculiarities of reaction are due, in the small gut, to the large formation of organic acids which more than counterbalance the slight alkalinity of the succus entericus, and, in the large intestine, to the output by the mucous membrane of an amount of highly alkaline secretion more than sufficient to neutralise the acidity (estimated by Gamgee to correspond to about 1 part of acetic acid in 1000 parts) of the intestinal contents as they pass through the ileo-cæcal valve.

Although the reaction of the intestinal contents is alone enough to account for the particular distribution of the intestinal flora, some share in determining the result must depend upon the physical conditions that obtain in the different parts of the gut. Thus, in the small intestine the absorption of fluid and the rapid transit of the food militate against any proliferation of bacteria that have passed through the pylorus without change. Indeed, the antagonistic forces are so considerable that the intestinal contents at the ileo-cæcal valve are, under normal circumstances, practically sterile.

In the *large intestine* the physical conditions are different from those which have just been mentioned. For, owing to the presence of sacculi and the much slower rate at which the intestinal contents progress along the gut, special facilities are given for multiplication of bacteria, with the result that the number of micro-organisms found in the contents of the large bowel increases rapidly on passing downwards, until it reaches to a total that may be as much as two millions per cubic millimetre of fæcal material.

According to Drs. Lorrain Smith and Tennant, however, this normal distribution of the intestinal bacteria can be disturbed with great ease. Thus, if a mild irritant, such as an intestinal worm, be present in a part of the ileum which is normally aseptic, the part becomes the seat of a localised, but enormous bacterial growth. At a

little distance above and below the situation of the worm the normal conditions obtain to a fair degree; but, if the irritation be widespread or long-continued, or both, the curve of bacterial growth becomes converted into one gradual but unbroken line of ascent from the duodenum to the rectum. Moreover, the condition which leads to this change may be very slight; anything which alters the character of the intestinal contents, and particularly anything which alters their reaction, may bring it about. This fact easily explains the very different results obtained when the investigation is made with the intestinal contents of cadavera.

The most important—as being the most commonly met with—of the intestinal bacteria are those which belong to the group known as the "colon group," B. enteritidis sporogenes (Klein), and B. lactis ærogenes. In the "colon group" are included a somewhat large number of bacilli that have many characters in common and also many differences, some of which are slight and others considerable. They all agree in being non-liquefying, short, motile bacilli, in forming gas in glucose media, in producing indol, in coagulating milk by the ferment which they form, in producing acid and reddening litmus media, in being asporogenous and strongly aërobic—although capable of multiplying under anaërobic conditions. But the various members of the group differ amongst themselves in the rate at which they form gas, indol, acid, etc., some showing the different reactions after one or two days' growth, others not showing them until a week or more has passed. But a matter of greater importance is that the members of the group show the greatest diversity in pathogenicity. Of two forms presenting identical characters in cultivation, one may be almost innocuous for the lower animals—such as the guinea-pig and the rabbit—whereas the other kills, on intra-peritoneal injection, within twelve hours. It is only if these facts are recognised to the full that it is justifiable to speak of the members of the group collectively as "bacillus coli communis." B. lactis ærogenes is not of any great importance so far as is known. It is not found largely in the intestinal contents of adults, but it is a normal inhabitant of the intestine of milk-fed infants. In this connection it may be mentioned that streptococcus coli gracilis, which is found in the meconium, though not in the intestinal contents of sucklings, is constantly present when a flesh diet is employed.

Of bacteria causing diseases of the intestines the most important are bacillus typhosus, vibrio choleræ asiaticæ, B. tuberculosis, B. enteritidis (Gaertner), B. dysenteriæ (Shiga, Flexner), actinomyces, and perhaps B. coli communis. A few points may be mentioned with regard to certain of them.

One of the most important problems engaging the attention of pathologists at the present time is that of the actual relation that obtains

between the typhoid bacillus and the various members of the colon group. These bacilli are not only very similar in their cultural characters, they are also extremely liable to be found together. It is not astonishing to find that the colon bacillus is present in the intestinal contents in cases of typhoid fever, but it is certainly remarkable that though in a large number of cases of the disease a pure culture of typhoid bacilli may be obtained from the spleen, in not a few cases of what is clinically typhoid fever cultures taken from the spleen give a mixture of typhoid and colon bacilli. It is possible, too, that a relatively pure culture of colon bacilli may be obtained from the heart's blood, while a pure or relatively pure culture of typhoid bacilli is obtained from the spleen. These points, and others of a similar kind, raise two important questions: (1) Is the typhoid bacillus a specialised variety of the colon bacillus, of which so many varieties are known? and (2) Does the association of the two varieties of bacillus cause the cases in which such association obtains to assume some special type?

With regard to the first of these questions, the most that can be said is that the prevailing view at the present time is that the two varieties are fundamentally distinct. The fact that there is the greatest similarity between the metals sodium and potassium is no reason for considering them to be identical substances, or that one is merely a variety of the other. This problem is itself intimately bound up with the further question as to whether the innocuous bacteria in the intestine can at times assume pathogenetic characters. We know, without doubt, that the virulence of bacteria generally may be exalted or diminished by variations in the conditions of their growth. A specific instance of this fact is afforded by members of the colon group, which, speaking broadly, are innocuous to lower animals when taken from healthy bowel, but are usually highly virulent if the bowel be diseased in any way, or if they have gained access to the peritoneal cavity. There is thus a probability that such bacilli of exalted virulence play a distinct part in causing the symptoms occurring in the person from whom they are obtained. But whether they can be the *causa causans* of a substantive disease or can only act as adjuvants is unknown.

With regard to the second question, the possibility at once arises that the colon bacillus is only present because, shortly before death, there has been an invasion of the blood by bacilli from the intestine, and that this is only a special example of a well-recognised fact. There is some reason, however, to consider that the mixture of infection in the case under consideration, as in the case of mixed infections in diphtheria, modifies the course of the disease, and probably in the direction of increasing its severity.

The matter has recently been taken a little further. It has been found that in certain cases of typhoid fever, cases which are generally of a somewhat less severe type, a micro-organism can be isolated which

differs in some particulars from B. typhosus on the one hand and B. coli on the other. These differences consist principally in the power of fermenting sugars. The subject is by no means fully worked out, but it has been suggested that there will have to be separated from typhoid fever a group of cases which is caused by a different bacillus, and which it has been proposed to indicate by the name "paratyphoid."

With regard to the affections of the intestinal tract, and particularly those of the appendix, that depend upon the presence of the ray fungus, it has now to be recognised as possible that they are not all caused by actinomyces, as is generally thought. It is now known that a number of the streptothriceæ besides actinomyces are the cause of disease, and as these fungi present very similar characters when found in the pus that they occasion, it is only after a prolonged examination of the characters of the observed micro-organism on cultivation that it can be named. When such cultures have been made in the case of mycotic disease affecting other parts, the original diagnosis of actinomycosis has often had to be changed.

The bacillus of dysentery, which was isolated from cases of the disease in Japan by Shiga, and was also found in acute cases occurring in America by Flexner and his pupils, has only recently been described, and must still be regarded as being on probation. There is the strongest reason for believing that although the bacillus in question may be definitely causal in certain cases of dysentery, this will not be so with regard to all. It is quite possible, even probable, that the causes of dysentery are many, just as the clinical varieties of the disease itself are many. With regard to Shiga's bacillus, indeed, it has hardly yet been conclusively proved that it is not a constant accompaniment of certain forms of dysentery rather than the actual cause. According to the recent work of Duval and Bassett (Baltimore), this same bacillus is the cause of the summer diarrhœas of infants. Their researches, however, have not been published long enough for confirmation.

Of B. enteritidis (Gaertner) it is only necessary to state that it is found in the intestine and in the blood and tissues in a large proportion of cases of food-poisoning. Only within the last few months (1902) Professor Delépine has traced to it an extensive epidemic originating in Derby, and occasioned by the consumption of infected pork pies and sausages. In some other cases of food-poisoning B. botulinus, a strict anaërobe, has been found, and in yet others the cause has apparently been Proteus vulgaris.

In the case of some other conditions affecting the intestinal tract and the parts in its immediate neighbourhood matters are even more obscure. Thus in the disease, or diseases, known under the name of "ulcerative colitis," the remnants of the intestinal mucosa are found to be crammed with bacteria of the most varied description. The great majority of them are, no doubt, accidental, but it is probable that

amongst them must be reckoned that which is the real cause of the affection in its early stages. Hitherto, however, the subject has not been much approached from its bacteriological side. With regard to appendicitis the position is much the same. Though Mr. Lockwood's observation that the submucosa of the appendix is very intimately connected with the sub-peritoneal tissue explains why affections of the interior of the tube should lead to peritonitis, the actual cause of the changes within the appendix itself are uncertain. At one time it was thought that the cause was the colon bacillus, but this view is now for the most part given up, and no other view has so far become paramount. Probably in the case of this affection, too, it will ultimately be found that it depends, not on one cause, but on several. The same is probably true of tropical diarrhœa or "sprue," from cases of which, however, Dr. Wethered has isolated a somewhat special bacillus.

Intestinal bacteria may find their way to other parts. This is typically the case with putrefaction after death, as has already been said. One need only advert, moreover, to the frequent occurrence of terminal infection of the blood. But even during life, and when death is not imminent, it is probable that such an invasion of other parts sometimes occurs. This is particularly likely to take place by way of the common bile duct, and it is probable that a considerable number of cases of cholecystitis originate in such a way. It is not impossible, either, that invasion of the gall-bladder by intestinal bacteria may be one of the causes for the formation of gall-stones. In the so-called "Pictou disease" of cattle, too, Adami has shown that the hepatic fibrosis that characterises that disease is intimately bound up with the presence of bacteria that have reached the liver by travelling up the common bile duct. And it is held by some that certain forms of human cirrhosis, especially that known as "biliary," may originate in the same way.

Although it is extremely doubtful whether bacteria can pass through the physiologically intact intestinal wall, there is no doubt that a distinct anatomical lesion is not necessary to permit of their exit. This is clearly shown in the case of strangulated hernia, for the blood-stained fluid in the hernial sac contains a considerable number of bacteria, particularly B. coli. Very similar is the case of abscesses in the neighbourhood of the intestines, but then in many instances it is impossible to be certain that the local condition has not been directly induced by an intestinal lesion. It is noteworthy in this connection that the contents of psoas abscesses are usually free from extraneous micro-organisms (often, indeed, from micro-organisms of any kind), and have no fæcal odour.

A point of great interest is the origin of the streptococci that are so generally found in the peritoneal cavity in peritonitis. Leaving entirely out of consideration those cases that depend upon disease of the female generative organs, even in those which supervene upon the occurrence of perforation of the stomach or gut, or upon the strangulation of the

bowel in a volvulus or other variety of intestinal obstruction, streptococcus pyogenes is the organism most commonly present. In these cases it cannot be doubted that the streptococcus has passed from the intestinal lumen to the peritoneal cavity, and yet streptococcus pyogenes is by no means a common inhabitant of the gut, and even if it gain access to the stomach it is so little resistant that it is not likely to persist far down. A still greater difficulty obtains in the case of primary pneumococcic suppurative peritonitis, unless it is supposed that the micro-organism is carried to the peritoneal cavity by way of the blood and lymphatic streams. And even this supposition would not explain the case of the streptococcus.

Antiseptic measures and the conditions that call for them.—It can easily be understood that the facts concerning the numbers and characters of the bacteria inhabiting and invading the alimentary tract which have been indicated above soon led to the question as to how they are best to be combated. These questions are summed up in endeavours to procure antisepsis of the tract in those various conditions which depend essentially upon undue multiplication of the bacteria in different parts. In the *mouth* there are decaying teeth, and the various forms of stomatitis from that relatively simple variety which constitutes aphthæ to the formidable variety constituting noma or cancrum oris. In these cases tooth powders and mouth washes of different kinds, particularly those which contain a small amount of carbolic acid or chlorine water, should be employed. Naturally in the case of infants with aphthæ, milder measures must be adopted, and then the tincture of myrrh is of use. When the teeth are carious, or the condition of pyorrhœa alveolaris is present, the measures adopted must be more vigorous, and must include a thorough treatment by the dentist. In all cases, however, and this must be held to obtain for all diseases of the kind under consideration, whether they affect the mouth alone or any other part of the alimentary tract, it is of the highest importance that the character of the food should be most rigidly supervised. In some cases farinaceous food, in others proteid, and particularly milk, should be given sparingly. It is impossible to give precise rules for the course to be adopted in any given case; the physician must be largely guided by circumstances.

With respect to the need for, and the methods of applying antiseptic measures to the *gastro-intestinal tract*, several considerations have to be borne in mind. That many of the symptoms, both local and remote, of affections of this region are the result of auto-intoxication brought about by bacterial agency in the canal appears to be certain; although it may be that this is not so potent or so general an etiological factor as has been supposed by some. Hence it would follow that antisepsis is in certain circumstances called for, whether this be to correct an excessive activity of what are normal processes in the stomach or bowels, such as

the immoderate fermentations of carbohydrates or fats; or to destroy such specifically pathogenetic bacteria as may have obtained entrance into the body, such as those of typhoid fever, cholera, and dysentery, or to prevent the possible assumption of toxic powers by previously innocuous organisms. The important effect of the surrounding circumstances, such, for instance, as the degree of acidity or alkalinity of the gastric or intestinal contents, the character of the food, and the mutual influence of various organisms in causing or intensifying toxic action, must also be taken into consideration, with any antiseptic treatment that may be proposed. Since the most extensive and varied bacterial activity normally takes place in the large bowel, without the production of untoward symptoms, it would seem very probable that some antiseptic or antidotal precautions are normally developed, on the break down of which disease may ensue; and it is at least possible that some of the drug measures employed to remedy the defect may fail in their anticipated effect, or may even increase the mischief, by interfering with this natural protection. Be this as it may, it is certain that the antiseptic treatment of the intestine is not so effective as might be expected on theoretical grounds. Further, also, owing to the extent of the bowel, few antiseptic drugs can be administered in sufficient quantity to be of use except at the risk of injury to the patient. At the same time a beneficial result may follow upon a treatment of this nature which stops short of being actually bactericidal, a completely aseptic condition being probably impossible of attainment. And, lastly, it is always uncertain how far a drug given by the mouth is decomposed, or absorbed, before reaching that particular part of the canal where its action is desired.

Next to such all-important prophylactic measures as the supervision of the food and avoidance of infection of the canal from unhealthy oral states comes the treatment of the stomach contents. Inasmuch as the hydrochloric acid of the gastric juice is inimical to a large number of organisms, pathogenic and other, any deficiency in this secretion must be corrected, not only for the purpose of arresting improper fermentations in the stomach, but also with the object of beneficially affecting the contents of the intestines by influencing the flow of pancreatic juice and in other ways. And a further and even more efficient correction of such unhealthy gastric states is lavage, which is a desirable supplement to other remedies in severe cases. Among the many drugs recommended for the treatment of flatulence and other evidences of undue gastric fermentations, such as are seen in their greatest degree in dilatation of the organ, may be mentioned:

Carbolic acid (gr. ½ to 1 in a pill, or ℞ Acidi carbolici, gr. i.; glycerini, ♏x.; sodii bicarbonatis, gr. xv.; aquam menthæ piperitæ, ad. ℥j. M. ft. haust.).

Creosote (1 to 5 ♏, or gradually increased up to ℥ss.; in capsule or emulsion, or as mistura creosoti, B.P., ℥ss. to ℥j.).

Sulphurous acid (ʒss. to ʒj. ; and the hyposulphite of soda, 10 to 60 grs.).

Sulphocarbolate of soda (5 to 15 grs. ℞ Sodii sulphocarbolatis, gr. x.; sodii bicarbonatis, gr. xv.; syrupi, ʒj.; aquam chloroformi, ad. ℥j. M. ft. haust.).

Salicylic acid (gr. 5 to 20).

Resorcin (gr. 3 to 8. It should be well diluted. ℞ Resorcin, gr. v. ; syrupi aurantii, ʒss. ; aquam pimentæ, ad. ℥iss. M. ft. haust.).

β-naphthol (gr. 3 to 10 in cachet. This is apt to be very irritating).

Menthol (gr. ½ to 2 in pill, or in chocolate tabellæ). Thymol (gr. ½ to 2, in pill or pastille).

The various salts of bismuth may also be included in this category. The officinal waters (dill, fennell, anise, caraway, peppermint, etc., and aq. chloroformi) are the most suitable vehicles for the foregoing when given in liquid form. Even more numerous are the drugs that have been employed in the antiseptic treatment of the intestine, and with varying benefit. Without doubt one of the most efficient methods is purgation, and unless otherwise indicated a full dose of castor oil or Epsom salts may remove in great measure the toxic agents, or expel some irritant substance which is producing an unhealthy condition favourable to the proliferation of bacteria. Small and frequently repeated doses of gray powder, calomel, or perchloride of mercury are without doubt the most effective for the desired purpose, and their aperient action still further increases their usefulness. Salol (5 to 15 grs.), resorcin, β-naphthol, benzo-naphthol (6 grs.), salicylate of bismuth (gr. 10 to 15) are among those most generally employed. To obviate the action of the gastric juice, drugs designed for intestinal antisepsis may be given in keratin capsules.

W. S. Lazarus-Barlow.

DISEASES OF THE MOUTH

The mouth is the seat of much traffic ; it is an air-way, the food channel, and the place where the first act of digestion is performed. Here, as elsewhere, complexity of function entails multiplicity of disease-processes. Of these such as arise in the bony skeleton belong strictly to the province of the surgeon ; certain others, such as the neoplasms of the soft parts, are amenable to surgical treatment alone, but as causes of ulceration it is necessary for the physician to acquaint himself with their diagnostic peculiarities.

The present state of pathology does not allow of a thoroughly scientific classification of the maladies which are about to be considered. Oral inflammations may be regarded in three broad and simple divisions, according to whether they affect the whole mucous membrane—*stomatitis ;* the tongue alone—*glossitis ;* or the gums only —*gingivitis*. Next the nervous affections of the mouth will be treated of, and then certain non-inflammatory affections, chiefly of congenital origin. Finally, the more important abnormalities of the salivary glands will come briefly under review.

It must not be forgotten that the mouth, besides being subject to its own peculiar disorders, is liable to be symptomatically affected in many general diseases. References to these oral symptoms will be found under the headings of the maladies to which they point, such as, for instance, measles, scurvy, varicella, and so forth (Vol. I.). Some remarks upon the tongue as an index of disease will be offered at the commencement of the section treating of its special affections.

STOMATITIS

The mucous membrane of the mouth may be the seat of the initial lesion in infective diseases, frequently in actinomycosis, sometimes in syphilis, rarely in tuberculosis and gonorrhœa. The variations from the normal type of these affections engendered by this peculiarity of situation are not of sufficient importance to warrant description here.

The following are the principal forms of Stomatitis proper. It should be understood that this classification aims more at clinical convenience than pathological exactitude, for mixed infections are

exceedingly common in the mouth, and some or other of the fifty odd oral microbes which have been described may at any time intervene to convert a simpler into a more complex type of the affection :—

1. Simple or catarrhal stomatitis.
2. Angio-neurotic stomatitis.
3. Infective stomatitis { *a.* due (probably) to bacteria; *b.* due to higher fungi.
4. Gangrenous stomatitis.

Simple or Catarrhal Stomatitis

This, the commonest form of inflammation of the mouth, is seen chiefly in children, though there is no period of life at which it does not occur.

Etiology.—The principal causes of catarrhal stomatitis are (*a*) mechanical, (*b*) toxic, and (*c*) specific (in general infections).

Among the mechanical causes the irritation of erupting teeth is the commonest. In infancy one has also to reckon with unclean and ill-fitting feeding implements. Burns and scalds of the mouth are at this stage of existence more frequent than in later life, when, however, the buccal mucous membrane is liable to be irritated by the abuse of tobacco, of stimulants, and of condiments, and by inattention to carious teeth. The dust inhaled in certain occupations, such as cotton-spinning, hop-picking, coal-mining, and paper-making, may also induce stomatitis. The toxic causes include poisons introduced from without, such as mercury, phosphorus, arsenic, lead, antipyrine, iodine, and bromine, and those developed within the body, as in uræmia and diabetes. The stomatitis so often seen in anæmic and leukæmic conditions may also be due to auto-intoxication. The principal general diseases which may be complicated by stomatitis are scarlatina, variola, measles, and varicella. Of foot and mouth disease the stomatitis is a chief diagnostic sign (see Vol. I.).

Fouquet, Galippe, and others hold that certain forms of catarrhal stomatitis spread by contagion.

Symptoms.—Simple stomatitis may affect the whole mucous membrane or be limited to a part of its surface. Its appearance is signalised by a dusky-red colour of the mucosa, together with a burning sensation, which is increased by any movement. The redness may be uniform or punctate ; it is accompanied after a while by swelling. The mouth is at first dry, but later on there is salivation.

The breath becomes offensive, and mastication, deglutition, and even speech may be painful; the discomfort occasioned by them is lessened on the appearance of salivation. The sub-maxillary glands are sometimes slightly swollen. There is loss of appetite with general malaise; the temperature is usually sub-febrile; in children there may be diarrhœa or convulsions. When the complaint is at its height the inside of the mouth has a whitish look from heaping up of the sodden epithelium. Erosions may now appear, or the symptoms of one of the severer forms of stomatitis may supervene. With appropriate treatment, however, the condition ordinarily subsides within a week. In a few cases (Brissaud, Galippe) it appears to have given rise to a general infection. In older subjects, and especially where the cause (such as the irritation of carious teeth or of excessive smoking) is not attended to, the stomatitis may become chronic; the whole mucous membrane is then greatly thickened and œdematous, showing the imprints of the teeth. Later on the necrosed epithelium may be rubbed off, leaving the papillary layer smooth. The salivation now diminishes and may even give place to xerostomia. In other cases simple ulcers may form, each with a pseudo-membranous floor and a surrounding zone of hyperæmia. The type of catarrhal stomatitis varies to some extent with its cause. Thus the mercurial form attacks the gums first, leads to profuse secretion of stringy or blood-stained saliva, and may cause deep ulcers or even periodontitis. In saturnine stomatitis the tongue is swollen and covered with mucus, the mouth is dry, and there is much thirst. In both these varieties there is great fœtor of the breath.

Morbid anatomy.—This does not call for detailed description. The cells of the affected mucosa are swollen and in various stages of granular and fatty degeneration, with a varying degree of hæmorrhage into the sub-epithelial connective tissue. Crypts are seen containing débris intermingled with the ordinary bacteria of the mouth. Special micro-organisms have been described, but their specificity has not been demonstrated.

Treatment.—Prophylaxis is obviously of the greatest importance; the measures to be adopted, such as, for instance, the hygienic care of the infant's mouth, can be readily inferred from the causes given under the heading of etiology. When the affection is established the cause must be ascertained, and, if possible, at once removed. Habits conducive to oral irritation are to be checked, while carious teeth will call for the file or the forceps. The rest of the treatment may be summed up in one

word—antisepsis. The mouth should be repeatedly washed out with boiled water and, from time to time, with a 2 to 3 per cent solution of hydrogen peroxide or of boric acid, or a 1-in-2000 solution of thymol. A most valuable mouth-wash is the following:—

℞ Thymol, gr. iij.
Acidi benzoici, ʒviss.
Tincturæ eucalypti, ʒiiss.
Aquæ destillatæ, Oj.
M. Fiat collutorium.

This should be used immediately after meals and also at frequent intervals during the day. Potassium chlorate and permanganate are popular, but less effectual remedies. A pigment for application with a camel's-hair brush is the following:—

℞ Acidi borici, gr. xxx.
Potassii chloratis, gr. xx.
Glycerini, ʒiij.
Succi limonis, ʒv.
M.

And a useful tooth powder is the following:—

℞ Acidi borici, gr. xl.
Potassii chloratis, gr. xxx.
Cretæ preparatæ, ʒj.
Magnesii carbonatis, ad. ℥j.
M.

To this a drop of otto of roses may be added.

Ulcers may require touching with strong iodine solution or even with chromic acid.

It is to be remembered that cases of catarrhal stomatitis are very liable to relapse; and it has been known to become chronic.

Angio-neurotic Stomatitis

Under this heading may be gathered a group of forms of stomatitis, for the most part somewhat rare, the cause of which may be in all probability referred to defective innervation. The following are the principal types which have been recognised; it will be seen that they show a general correspondence with certain skin eruptions: (*a*) perforating ulcerative, (*b*) herpetic, (*c*) pemphigoid, (*d*) urticarial, (*e*) lichenoid.

(*a*) *Perforating ulcers* may appear in the mouth during the course of tabes. They are always situate on the alveolar process, are usually chronic and painless, and may lead to loss of teeth.

(*b*) *Herpes zoster* may attack two regions of the mouth, following the distribution of the superior and inferior divisions of the trigeminal nerve respectively. In the former case the vesicles appear in the mucous membrane of the cheek, gums, and pharynx of the affected side; in the latter they are situate on the part of the tongue supplied by the lingual nerve. The treatment is general; if there be much pain cocaine (2 to 5 per cent solution) may be given in a mouth-wash, and if the vesicles ulcerate they should be touched with 10 per cent silver nitrate solution. Arsenic internally is usually indicated.

The so-called herpes buccalis (properly stomatitis herpetiformis) is much more common. The vesicles in this case, which usually spread over the whole of the mouth and pharynx, and may involve the larynx, appear in most cases as the result of the administration of drugs, such as tuberculin and antipyrine, or in the course of infective disorders (pneumonia, influenza, and malaria). The affection lasts from ten days to two weeks (Kraus). The local treatment comprises ice to suck and astringent gargles.

(*c*) The pathology of *pemphigus* is so unsettled as to render a scientific account of the bullous affections of the mouth at present impossible. All the main varieties of pemphigoid eruptions have been described as occurring in the mouth. The most common form is that described by Rosenthal as erythema bullosum. It occurs mainly in middle-aged people. Large blebs appear all over the oral mucosa; a similar eruption is at the same time seen on the genitals, and sometimes on the mucous membrane of the nose and arms. The external skin escapes. The bullæ may suppurate and lead to chronic ulcers, but usually soon pass away, though they are extremely likely to return. There is no special treatment. When the bullæ of pemphigus proper make their first appearance in the mouth the disease is apt to run a virulent and often fatal course. This is especially true of the disease described by Neumann as *pemphigus vegetans*, where the oral eruption is followed after a week or ten days by crops of cutaneous blebs, which run a chronic course with much ulceration and papillary overgrowth. A large proportion of such cases ends fatally.

(*d*) *Urticaria* occasionally invades the mouth, leading to great œdema, especially of the tongue. The treatment consists in brisk purgation.

(*e*) The plaques of *lichen ruber* are sometimes noted in the mouth. They affect chiefly the buccal mucous membrane, but have also been described on the tongue. The treatment is by the administration of arsenic, with perchloride of mercury as a local application.

Infective Stomatitis

Under this head we have to consider certain acute diseases of the mouth, for which a microbial cause has actually been discovered, or is by analogy rendered extremely probable. These affections fall into two groups: in the first, comprising aphthous stomatitis, ulcerative stomatitis, and phlegmonous stomatitis, the evidence points to hitherto undetected bacteria as the excitants; the members of the second, of which thrush is the most important example, are associated with the constant presence of fungi, belonging to higher groups than the schizomycetes.

A. *Infective Stomatitis of probably Bacterial Origin*

i. Aphthous[1] or follicular stomatitis.—This name was formerly applied to many oral disorders, but is now restricted to the particular affection about to be described. It should be added that for most French and many German authors aphthous stomatitis is synonymous with foot and mouth disease in the human subject.

Etiology.—The disease occurs mainly in children, especially in the second year of life, and its appearance is certainly favoured by unhygienic conditions, more particularly by improper feeding. But it may be met with in adults whose teeth have been neglected. The cause in these cases may be dental irritation, excessive smoking, or the use of certain tooth-powders. Dyspepsia may lead to a very chronic form, and the affection may be bound up with sexual conditions. Thus aphthous stomatitis is not infrequent in pregnant women, while instances are on record in which it has recurred with every menstruation. The history of the onset resembles that of an acute exanthem and suggests a specific cause. As has already been said, the multitude of oral bacteria renders the determination of the part played by any one of them in a particular disease a matter of the greatest difficulty. In aphthous stomatitis the diplostreptococcus described by Stooss appears to be the most likely of the causative micro-organisms hitherto put forward. Lastly,

[1] Aphthæ and aphthous, from ἅπτειν, to inflame.

some observers hold that the affection is not a specific one at all, but that the aphthæ are merely complications of a simple stomatitis.

Symptoms.—The characteristic sign is the appearance of the so-called aphthæ on the oral mucosa. These are at first grayish or yellowish specks which may occur all over the mouth, but are chiefly seen within the lower lip. They grow after awhile into little plaques or papules; then their epithelial covering is rubbed off and erosions remain, each surrounded by a tiny zone of hyperæmia. The creamy-looking floor of the erosion is formed by sodden desquamated epithelium; the vesicular stage described by French writers is not commonly seen in England. The aphthæ occur singly or in groups, and come out in successive crops. They give rise to sharp, pricking pain, comparable to that of conjunctivitis; this is increased on movement and may interfere with swallowing. There is usually much fœtor of the breath. In favourable circumstances the erosions heal by overgrowth of epithelium from the edges within a few days, and the patient is well in a week or two. They may, however, relapse or pass on into chronic intractable ulcers. Aphthous stomatitis is in rare instances complicated by the appearance of a similar affection on the female genitals (Neumann); this runs an innocuous course, the only danger being that of confusion with syphilis.

Morbid anatomy.—Henoch has shown that the essential lesion is a fibrinous exudation in the superficial layers of the mucosa. The rubbing-off of the epithelium is due to mechanical causes; but there is probably also some local necrosis. The coagulation of the fibrin prevents the formation of ordinary pus.

Treatment.—Prophylaxis, by rigid attention to the hygiene of the mouth, is of course the first consideration. Unless the attack is severe and painful local measures are hardly necessary. Tobacco, alcohol, hot and irritating foods, are of course to be forbidden. The best mouth-wash is a 1 per cent solution of boric acid; potassium chlorate is often painful, and a little opium may have to be added to the wash containing it. A weak solution of cocaine may be used (2 per cent in children, 5 per cent in adults) if there be much pain. It is sometimes necessary to touch the aphthæ with a fine-pointed silver nitrate pencil; or they may be painted with a solution containing 1 per cent sodium salicylate and 2 per cent cocaine in distilled water.

Bednar's aphthæ.—This affection is almost confined to the new-born, and is characterised by the appearance of aphthous plaques (plaques pterygoidiennes) on the gums and soft palate.

The researches of E. Fraenkel appear to show that it is a microbial ulceration in which the ordinary pus-cocci play the most important part. The prophylaxis and treatment should follow the above-mentioned hygienic lines.

ii. Ulcerative stomatitis.—Ulceration may, as has already been said, complicate any simpler form of stomatitis, but the affection next to be considered is marked off by its characters and course as a distinct clinical entity.

Etiology.—The disease is most common between the fourth and eighth years of life. In adults it has most often been observed in armies, sometimes in workhouses, ships, and prisons. It has frequently occurred in inmates of foundling hospitals and orphan asylums; it assumes sometimes endemic, sometimes epidemic characters. Of its contagiousness there can be no doubt, for Bergeron succeeded both in inoculating himself and in communicating the disease to others. Its actual exciting cause is not, however, so certain. One of the most potent predisponents is undoubtedly the presence of teeth, for it occurs neither before their appearance nor after their loss; the accumulation of tartar accentuates the liability. Conditions of depressed vitality, such as rickets, diabetes, and scurvy, lend their aid; local necrosis supervenes, and the ever-ready oral bacteria complete the process. Whether the disease is pathologically specific remains to be seen, for no single causative micro-organism has thus far been detected; of those found in the floor of the ulcers, spirilla appear to be the most constant.

Symptoms.—The ulceration always first attacks the gums, commencing in the little trench at the base of the (usually molar) teeth. In adults the erupting wisdom teeth are the common centres of the disease. There is at first swelling of the affected region, which assumes a dusky red colour and is intensely painful. The swollen gums are spongy, and, as a rule, wholly, or in part, covered with a yellowish brown pultaceous crust of desquamated and sodden epithelium which can be easily detached, laying bare the sensitive and often already ulcerated mucosa. Within three to five days ulceration is definitely established. This commences in the part first affected, usually in the lower jaw; the ulcers have jagged, swollen edges and yellowish bases, which look as if they were covered with sodden membrane. They extend along the alveolar margin, and in severe cases affect the cheeks, the lips, the palate, and the tongue. There is usually œdema of the surrounding parts, which are not, however, indurated. This œdema may extend to the pharynx and cause dysphagia. From the beginning there

is salivation, and the breath acquires a disgusting odour, which has led to the application of the name of "putrid sore mouth" to the disease; the stench reaches the acme of offensiveness when the quasi-membranous covering of the ulcers has become converted into a grayish or blackish brown slough. The general symptoms are, as might be expected, loss of appetite, malaise, and slight fever. The sub-maxillary gland is swollen on the affected side. The normal course of a properly treated case is now favourable. A zone of demarcation appears round the ulcers, and the process of repair follows its regular march. The cheeks, tongue, lips, and palate recover before the gums, which are always more deeply affected. Sometimes, however, and this is particularly the case in mercurial stomatitis, the ulceration spreads inevitably to the deeper parts, attacking the tooth sockets and the maxillary processes, and leading to necrosis of the jaw and septic infiltration of the connective tissue planes of the neck. When the mercurial cachexia is added to these alarming extensions death almost always results; even when this complication is not present fatal septicæmia may supervene. But the prognosis of ulcerative stomatitis is in general favourable as regards life, though the local affection may persist, with relapses, for weeks or months. Noma occasionally appears as a complication or sequela of the utmost gravity.

Treatment.—The occurrence of the disease can no doubt be prevented by keeping the mouth and teeth clean, avoiding the excessive use of mercury, attending to local sources of irritation, and looking after the general health. The gums may be hardened by the use of some such mouth-wash as that recommended by P. J. Pick, viz. menthol, two parts, alcohol and tinct. krameriæ, of each one part. When the disease is established astringent mouth-washes are indicated; five grains of alum and fifteen of potassium chlorate in an ounce of water form a most valuable combination. The latter salt is indeed almost a specific for the disease; it may be dusted over the ulcers and a few lozenges should also be sucked daily. The severer and more chronic forms of ulcerative stomatitis are extremely refractory to treatment. Most benefit in such cases follows from picking off all crusts and then applying iodoform to the raw surface. It need hardly be added that the state of the general health requires most careful attention.

iii. PHLEGMONOUS STOMATITIS.—This name has sometimes been used to imply the disease just described, but should be reserved for a somewhat rare affection which is related to erysipelas on the one hand and Ludwig's angina on the other.

Etiology.—Streptococci have been found in virtually all the cases examined, but whether they are identical in every instance with the erysipelas-coccus of Fehleisen is still under discussion. If not it must still be admitted that this micro-organism is sometimes the cause of phlegmonous stomatitis, which may indeed complicate facial erysipelas. As in all such affections, it is immaterial whether the cocci enter through a gross lesion or a microscopic abrasion.

Symptoms.—There is rapid, dry swelling of the oral mucosa, particularly in the floor of the mouth. Much œdema develops, there is intense pain, the temperature rises, and the patient is profoundly ill. The tongue is especially swollen. The affection may spread to the larynx; the pharynx is almost always involved. As the disease advances diffuse suppuration occurs in the deeper layers of the inflamed structures. There is considerable danger to life; the patient may suffocate or succumb to toxæmia.

Treatment.—Little can be done beyond keeping the mouth clean with astringent and antiseptic washes, giving ice to suck, and if necessary incising the most swollen parts. Tracheotomy may be called for. General tonics are urgently indicated and stimulants may be used freely.

B. *Infective Stomatitis due to the higher Fungi*

i. Thrush—**Etiology.**—This disease is commonest in infancy; in adults it is usually a terminal complication of some slowly lethal condition, such as old age, phthisis, and cancer. Thrush may be described as catarrhal stomatitis, complicated by the presence of a special fungus. The generic position of this fungus is not yet certain. It was long regarded as a hyphomycete, and received the name of oïdium albicans; of late the opinion appears to be gaining ground that it belongs to the group of blastomycetes or yeasts, and that its name should be saccharomyces albicans. This is the view adopted in the valuable monograph of Guidi (*Mughetto, Micologia et metastasia*, Firenze, 1896); it is not, however, easy to explain the mycelium-like threads on this hypothesis. The fungus is readily cultivable, and shows considerable pleomorphism on different media; in the actual disease it appears to live symbiotically with various bacteria. It does not grow readily in fresh milk, but multiplies freely when this is stale. Its appearance in the mouth is favoured by unhygienic conditions, especially dirty feeding-bottles, and by anything preventing the normal cleansing action of the lingual

movements. It has apparently been transmitted from one child to another through the intermediation of the nipple.

Symptoms. — The development of the classical picture of thrush is almost invariably preceded by some catarrh of the oral mucosa. The seats of election are the tip and edges of the tongue, and the labio-mandibular fold. These become covered with pearly specks, rapidly increasing in size and soon coalescing into a whitish membrane, much resembling the "skin" of boiled milk. The mouth is soon extensively involved, and in children, at any rate, there is a good deal of pain. The fungoid membrane can be readily scraped or wiped away, but is not sufficiently coherent to be peeled off. The subjacent mucous membrane is red and subacutely inflamed; there may be some epithelial desquamation, particularly of the papillæ. If the disease become chronic the membrane may be felt-like in consistency, overlying a smooth, hard, injected mucosa. Thrush varies very much in severity. It may clear off spontaneously in three or four days, or persist and increase till the infant, worn out by inability to suck, dies of some intercurrent, usually intestinal disease. In adults, as has already been said, it is mainly a terminal complication, but it may occur in cases of typhoid, which eventually recover. The fungus rarely penetrates the deeper layers of the mucosa, but may in isolated instances enter the circulation and cause embolic abscesses in the brain or kidney. Sometimes it involves the pharynx and even spreads down the œsophagus. It occasionally attacks the external genitals, and is said to have been found in the urinary bladder. The popular expression that "the thrush went through the child," probably connotes no fact of greater pathological significance than that a child with a dirty mouth is likely to have an unclean anus. Healing results from extensive epithelial exfoliation, which entails shedding of the characteristic covering.

Morbid anatomy.—The membrane consists of the spheres and threads of the fungus, together with bacteria, epithelial scales, and a few leucocytes. Fibrin is absent from it. Ulceration of the underlying parts is extremely rare. The secondary emboli contain the characteristic threads.

Treatment.—Prophylaxis is here again of primary importance. The sterilisation of milk and the cleaning of nipples and feeding-bottles is essential. In adults, when the mouth remains open in diseases such as typhoid, it should be repeatedly cleansed; for which purpose a suitable mouth-wash is made by adding tinct. myrrhæ, ℥xx., and tinct. krameriæ, ʒj., to a wineglassful of warm

water. When the fungus has established itself it should be wiped off with a soft rag soaked in boracic acid solution, and the exposed surface dabbed with solution of perchloride of mercury (1 in 4000), or borax (10 grs. to the oz.). Foods and drugs containing sugar should be prohibited, as the fungus grows very readily on saccharine media. The general health usually requires attention, and even in children it is often advisable to give alcohol.

ii. Leptothrix mycoses of the mouth.—The normal fungi of the mouth are leptothrix buccalis, spirochæte dentium, and spirillum sputigenum (Kraus). Of these the first-named is a constant saprophyte in health, and its growth is favoured by most pathological conditions, which reduce the resisting power of the epithelium. It is found in abundance in connection with dental sordes, tartar, and caries, with salivary calculi and with tonsillar concretions. Whether its association with these is causative or merely casual remains to be determined. It certainly seems to play an important part in the pathogenesis of the condition known as pharyngomycosis leptothricia (p. 122), which may invade the palate and gums from behind. It has been found in abscess of the tongue and frequently in suppurative tonsillitis. Its association with these is probably secondary.

iii. Mycosis sarcinica.—Sarcina oris is not certainly a distinct species. It is found in the mouth in marasmic individuals, particularly in connection with inflammatory conditions. It may occur in health.

iv. Mycosis mucorina.—Conditions allied to "black tongue" (p. 89) have been described on the gums and cheeks. Mucor niger attacks chiefly the tongue; aspergillus may spread in from the nasal cavities, and one case is recorded by Paltauf in which, apparently, mucor corymbifer led to phlegmonous pharyngitis, and eventually fatal pyæmia.

v. Actinomycosis.—The oral manifestations of this affection will be found described in Vol. I. (p. 246).

Gangrenous Stomatitis

Syn. Noma, Cancrum Oris

Noma is, of course, strictly speaking, a form of infective stomatitis, but its special characteristics, and in particular the fact that it may supervene upon any of the other varieties of stomatitis, seem to entitle it to a separate consideration.

Etiology.—Noma occurs almost exclusively in children; it is a rare disease, and is fortunately becoming more and more infrequent. A few cases have been recorded in adult life (even up to seventy years of age), but fully 95 per cent of patients are under twelve years old. It is extremely rare for noma to occur as an "idiopathic" disease in previously healthy subjects; its victims are as a rule already exhausted by some antecedent affection. Of these by far the commonest is measles; next, perhaps, comes typhoid fever (an especial precursor in adults), and then scarlet fever, pneumonia, whooping-cough, and malaria. Chronic diseases, such as tuberculosis and syphilis, may also precede the affection, which may furthermore, as has already been said, supervene upon ordinary ulcerative stomatitis.

No micro-organism has as yet been proved the specific cause of noma. The relation to the disease of the rods described by Lingard, Babès, and Schimmelbusch is undetermined. The bacillus described by Grawitz closely resembles that of calf diphtheria, and Bang and Loeffler have endeavoured to show that both affections are due to it. Freymuth and Petruschky, and quite recently Walsh of Philadelphia, have described the constant occurrence of the diphtheria bacillus in noma. Other observers find only the microbes of pus infection. Generically the disease appears most closely allied to hospital gangrene; in both it is possible that conditions, external or internal, which are unfavourable to the patient, may lead to the exaltation of the virulence of a micro-organism which is ordinarily of much slighter activity.

Symptoms.—Noma usually commences with the appearance of a vesicle on the mucous membrane lining the cheek opposite the first or second molar tooth. A few cases are recorded in which it began on the outside of the cheek, and at least one in which it took origin as a deep-seated phlegmon. It is usually limited to one side, and the left is said to be the more frequently affected, but both cheeks have been known to be involved. There may or may not have been salivation and the other symptoms of antecedent stomatitis. The vesicle is of a purplish colour, but soon becomes converted into an ulcer covered by a black scab; there is almost from the first a disgusting fetid odour. The cheek and lower lid swell with extreme rapidity; they are pallid in appearance and brawny in consistency. As the necrotic process in the mouth extends the visible swelling involves the surrounding parts with great speed. Quickly in the midst of the greasy-looking tumour there appears a reddish violet spot, which spreads in all directions, pushing before it a surrounding

zone of hyperæmia. As the circumference of this area extends, its centre, which corresponds to the site of the primary intraoral lesion, blackens, and the characteristic aspect of spreading gangrene is established. It is a striking fact that there is little or no pain at this or any other period of the disease. The patient, who has as a rule been feeble from the first, is now evidently gravely ill from septic intoxication. Still, it is remarkable that the child's general condition and spirits may remain apparently good, and it may play about and even eat as usual till within a few hours of its death; a similar anomaly is sometimes seen in septic peritonitis. But grave cases of noma, and the majority fall within this category, have almost always hectic fever, the temperature often rising to 104° or higher. In such instances the rapid course continues; the gangrenous process attacks the bones of the face and jaws; the swelling spreads to the neck and scalp; and the child dies in coma, often preceded by delirium. The most frequent complication is septic pneumonia, which is, of course, of the aspiratory or lobular type; diarrhœa is also very common, and there may be gangrene of other parts, such as the lungs or stomach. Occasionally the facial artery may be ulcerated into, with fatal hæmorrhage. Noma of the ear or female genitals may also occur, sometimes in epidemics, but not necessarily as a complication of the face affection.

The prognosis is grave, over 70 per cent of cases terminating fatally. When spontaneous recovery occurs it is accompanied, as in other forms of gangrene, by the establishment of a zone of demarcation round the diseased area.

Morbid anatomy.—The microscopical appearances are those of moist gangrene; in the centre is a slough which is surrounded by a ring of thrombosed blood-vessels. It is characteristic that there is little or no reactive inflammation, at any rate in fatal cases. Infiltration is scanty, the tissues appearing to die and liquefy without any preceding vascular changes.

Treatment.—To have a chance of success this must be prompt and vigorous. Applications of fuming nitric acid are of but little use. The affected area should be at once excised without consideration for future deformity. If the bone is attacked it must be scraped; I have seen recovery in one case in which the malar bone was involved. The walls of the operation cavity are to be treated with the strongest antiseptics; its area may be extended beyond the visible limits of disease by Paquelin's cautery, or it may be plugged with lint soaked in a 40 per cent solution of zinc

chloride. It may be useful to inject glycerine of carbolic acid round the edges of the swelling.

At the same time the general health of the patient must be supported by every possible means. Alcohol, quinine, iron may be freely given, and the child should have plenty of fresh air and, if possible, sunshine.

Sokolow claims recently to have cured a case of noma by the use of red light. Improved hygienic conditions form the basis of prophylaxis and appear to be contributing to the disappearance of the disease.

GINGIVITIS

Inflammation limited to the gums is not very common. It may, however, occur during the process of eruption of the teeth, and may at the time of appearance of the wisdom teeth give some trouble. In debilitated individuals an infective gingivitis can develop, but the symptoms of this are soon merged in those of general stomatitis.

A well-marked form of gingivitis is that occurring in women in connection with menstruation or pregnancy. The gums are hyperæmic and bleed with great readiness; a slight irritant will cause ulceration, which may involve the dental periosteum, but is usually mild in character. A potassium chlorate mouth-wash is in most cases almost specific; at the worst the affection subsides spontaneously when the sexual ailment passes off.

Two other diseases of the gums require some particular mention —pyorrhœa alveolaris and gumboil.

Pyorrhœa alveolaris (Rigg's disease).—This complaint is commonest in adults and attacks women more often than men. Its cause is unknown; it is often thought to be due to irritation by masses of tartar, but it certainly may occur in mouths which are kept scrupulously clean.

The onset is insidious, and the first symptom is generally slight loosening of one or two teeth, usually incisors or anterior molars. On investigation there is found between the neck of the tooth and the gum a small cavity containing pus. Further examination shows that this condition is far more extensive than the symptoms indicate. The edge of the gum may be riddled with pockets, which threaten the existence of the whole denture. There is not often general stomatitis; the breath may be offensive, and there is sometimes a sanguino-purulent expectoration which suggests the suspicion of lung mischief. The course is chronic, and in the absence of

treatment many teeth may be lost. The remedy is to incise the gum over each pocket so as to expose the little cavity, which is then rubbed in with iodoform or repeatedly washed out with peroxide of hydrogen solution. Great patience on the part of operator and subject is required. Sound teeth should not be removed in this disease, but accumulations of tartar must be scraped off.

Gumboil.—This form of alveolar abscess, which usually arises in connection with a carious tooth-fang, is of chief importance as a not infrequent cause of reflex convulsions in children. It should be treated by free incision.

A curious condition, apparently incurable, of *hypertrophy of the gums* occasionally occurs in children, particularly those of weak intellect.

Note on the hygiene of the mouth in children.—It is well known that inattention to the teeth in adults is a fruitful cause of gastric troubles. Still more is this the case in children, who are also, as already seen, exceedingly liable to inflammatory affections of the mouth itself. Children's teeth should be brushed after every meal with a small badger-hair brush which has been dipped in warm water and then rubbed on pure curd soap. A little precipitated chalk may also be used. Excessive consumption of starchy and saccharine foods, particularly when the child is in bed at night, is to be checked. It has been abundantly shown that lactic acid is formed in the mouth by the action of micro-organisms on sugar.

DISEASES OF THE TONGUE

The tongue as an index of disease.—Before proceeding to consider the specific diseases of the tongue it will be advisable to give some attention to the appearances of the organ which are of value in the diagnosis and prognosis of general disorders.

In health the tongue should be firm, of medium size, and rounded at the tip; it ought to be under perfect voluntary control, and should show no tooth-marks. Its normal colour is pink, the papillæ being of a slightly deeper tone. A slight coating of yellowish fur is natural, particularly in the morning, when the tongue has for some time rested from the cleansing action of its movements. This fur is composed of bacteria mingled with the débris of desquamating epithelial cells, which chiefly invest the filiform papillæ, the fungiform remaining for the most part free. In infants the fur

is never thick, since the filiform papillæ are very small and but slightly developed. In certain pathological states the fur is much increased, partly from an actual multiplication of its elements and partly from their non-removal. As the bacterial portion is replenished from the surface and the epithelial from below, the influence of attrition is chiefly exerted upon the former; this fact, and not the cellular increase which has been supposed by some, is responsible for the preponderance of the epithelial element when the coating is unusually thick.

It has long been recognised that the condition of the tongue points rather to constitutional affections than individual diseases. Dr. W. H. Dickinson has, though his pathogenetic explanations may not be generally accepted, done useful service in classifying the morbid states of the organ and enumerating their relations to general diseases. He points out that the two chief influences in these lingual alterations are pyrexia and diminution in the secretion of saliva. The fur being normally most abundant upon the filiform papillæ, a slight exaggeration of this gives rise to the appearance designated *stippled*, while in the ordinary *furred* tongue the coating has spread equally over the whole surface. Such conditions connote a mild degree of pyrexia and slight inappetence, the secretion of saliva being fairly abundant. If the fur continues to increase in amount the tongue appears to be, in Dr. Dickinson's phrase, *plastered;* the fur loses its whiteness and moisture and becomes brown and dry. The cause is here continued fever with deficient salivary secretion; the result is well seen in severe infections, such as pneumonia and acute rheumatism. In scarlet fever the hyperæmic fungiform papillæ protrude through the creamy fur, giving rise to an appearance which a misguided attempt at picturesqueness has likened to that of a strawberry. If the saliva is lessened from the first the "plastered" appearance may not be seen, but the tongue may be covered with shaggy brownish patches, consisting of crusts attached mainly to the filiform papillæ. Either this or the plastered tongue may pass on into the *encrusted* condition, so familiar in typhoid; the tongue is now covered with a fairly uniform, blackish brown layer, seamed by irregular cracks and crevices. A similar deposit is seen on the teeth and receives the name of sordes; it is very largely composed of bacteria, moulds, and their products. Finally this crust may scale off, leaving a red, dry surface; this *bare* tongue is of ill omen, denoting, as it does, extreme prostration and absence of salivary secretion. The normal cleaning of an encrusted tongue begins

from the tip and edges; it is less uniform than the process of coating, so that the fur comes to assume a rough edge bordering a clean, moist surface. Valuable as they are, the diagnostic indications to be derived from the tongue have not now the same utility as when other means of investigation were undeveloped. Curiously enough these indications are most apt to be fallacious in diseases of the digestive system; a man may have a disordered stomach and a clean tongue, or even more frequently a dirty tongue with a healthy stomach. But the value of lingual inspection in prognosis remains; the changes occurring in an encrusted tongue will point almost unerringly to the patient's fate. As an index to treatment the tongue is also entitled to notice; the dry tongue betokens an anorexic subject and calls for fluid nutriment, the bare tongue for the vigorous exhibition of stimulants, the flabby tongue for tonics. But the custom of "treating the tongue" has, it is to be feared, been but too often a cloak for carelessness in the proper physical investigation of disease.

Glossitis

Our knowledge as to the pathogenesis of lingual inflammations is no more satisfactory than in the case of general oral infections. A classification of glossitis must hence be in the main morphological. We shall confine our attention to the forms which take their origin, and are as a rule confined to the tongue; as has already been stated the lingual mucosa participates in all ordinary varieties of general stomatitis. For the sake of completeness and, in particular, as an aid to diagnosis, the term ulcerative glossitis has been made to include all common forms of ulceration of the tongue, however originating. The description of such kinds as are mainly of surgical interest has been compressed, however, within the smallest possible compass.

The following classification offers a simple arrangement of the inflammatory affections of the tongue:—

1. Parenchymatous glossitis.
   a. Acute; *b.* Chronic.
2. Superficial glossitis.
   a. Acute; *b.* Subacute or neurotrophic; *c.* Chronic.
3. Ulcerative glossitis.

PARENCHYMATOUS GLOSSITIS

a. Acute (phlegmonous).—Acute inflammation of the substance of the tongue is rare; it most frequently attacks young male adults, and in the summer rather than the winter.

Etiology.—The most important predisposing causes are general ill-health and recent alcoholic excess; among excitants may be mentioned exposure to cold, trauma, mercurial stomatitis, acute infections, such as erysipelas and scarlet fever, and occasionally the stings or bites of insects.

The micro-organisms known to participate in the causation of the disease are strepto- and staphylococci. The presence of the former is associated with much œdema of surrounding parts, and may eventually lead to the condition known as Ludwig's angina. Staphylococci are more likely to cause pus formation, and may even lead to the development of an acute abscess of the tongue. It has been suggested that acute phlegmonous glossitis is often secondary to lingual tonsillitis.

Symptoms.—The condition appears with extreme rapidity. After a brief period of pain in mastication or deglutition the tongue suddenly swells up, and in a few hours occupies the whole mouth and may even protrude from it. The teeth are driven into the swollen organ and fixed so that speech and the deglutition of solids are alike impossible. There is usually intense pain; the saliva is secreted to excess and dribbles from the opened mouth. The temperature is always raised, but does not as a rule exceed 101°. The tongue is livid and covered with a creamy fur, which becomes brown, dry, and cracked where exposed to the air. Within twenty-four hours the disease is usually at its height, and the patient may be quite dyspnœic. The ordinary tendency is now towards amelioration, the swelling subsiding within the course of a week; small sloughs may appear on the surface, which scale off, leaving little ulcers. Suppuration is not very common, but may lead to chronic phlegmon or to septicæmia. Gangrene of the tongue may also follow; this has been described likewise as the result of syphilis and of malaria; it presents all the characters of sloughing phagedæna, and may be fatal. Other causes of death in acute phlegmonous glossitis are pneumonia and septic intoxication.

Hemiglossitis.—A similar acute process may affect only one-half of the tongue. It is less severe than the bilateral disease, and is usually accompanied by the formation of a definite lump in the

body of the organ. The left side is perhaps chiefly attacked, as the more liable to trauma in right-handed persons. Hemiglossitis is sometimes apparently of nervous origin, being then usually associated with facial herpes.

Treatment.—The measures required vary, of course, with the severity of the disease. A slight case needs, as a rule, nothing more than a brisk purge and slop diet for a few days. If the swelling be extreme, threatening suffocation, free longitudinal incisions are to be made on either side of the raphe. Tonics are, as a rule, indicated. Vigorous local antisepsis is called for if gangrene supervenes; abscesses will, of course, need to be opened. Tracheotomy is seldom required.

Sublingual phlegmon.—This rare affection attacks the connective tissue surrounding the sublingual gland. It produces pain and swelling beneath the tip of the tongue, which may become incapable of being protruded. The temperature is raised and there is salivation. The treatment is deep incision and drainage.

b. Chronic.—This is of but little importance; a nodule of persistent inflammation may follow acute (especially hemi-) glossitis, or may appear spontaneously as a tense, elastic swelling, in size up to that of a pigeon's egg, usually near the tip of the tongue. There is no ulceration. Such a condition may occur in advanced phthisis and pass on into the *cold abscess of the tongue.* Treatment consists of incision and curetting (Kraus).

The tongue is occasionally the seat of *actinomycotic infection*, which may lead to abscess formation (see Vol. I. p. 246).

SUPERFICIAL GLOSSITIS

a. ACUTE—i. *Membranous or pellicular glossitis.*—Diphtheria may very rarely commence as a glossitis. The diagnosis would depend upon the bacteriological examination and the clinical course of the case. Another form of membranous glossitis, associated with the presence of strepto- and staphylococci, is also described. The membrane may in these cases spread over an operation wound or follow one of the acute specific fevers of childhood, such as measles or scarlatina, particularly, it is said, where the patient is already the subject of impetigo of the face or scalp. In adults it may supervene upon exhausting diseases or occur in the mouths of excessive smokers. The treatment is to detach the membrane, which may leave a raw surface, and to paint the exposed parts with an antiseptic solution, such as 1 in 1000 corrosive sublimate.

ii. *Acute papular glossitis.*—This commences suddenly, with fever and pain in the mouth. Within twelve to twenty-four hours the tongue is covered with pale papules, each the size of a pea, set in a dusky-red areolæ of inflammation. The papules are due to inflammatory exudation in the deeper layers of the mucosa and in the submucous tissue. They become umbilicated and then slowly diminish, giving place to small erosions. There is some fœtor and often marked gingivitis. The etiology is unknown, and the treatment, according to Michelson, is to wash the mouth out with 4 per cent solution of borax.

iii. *Lingual papillitis.*—This is an intensely painful affection associated with minute erosions of the tips of the filiform papillæ. It is very rare and is treated by application of the galvano-cautery, or better by excision of the papillæ. Relapses may occur.

In children two other forms of acute superficial glossitis are sometimes seen. The one is an erythematous inflammation of the margin of the tongue due to the irritation of an ill-fitting teat; the other a vesiculo-pustular eruption of unknown origin, but probably spread through milk. The treatment in either case is to remove the cause, if ascertainable, and to apply antiseptics.

b. Subacute or neurotrophic.—This corresponds roughly to the angio-neurotic group of general inflammations of the oral mucosa already considered. The members of each class are allied more by etiological uncertainty than by generic similarity.

i. *Geographical tongue* ("wandering rash of the tongue," etc.).—This disease is somewhat uncommon; it is much rarer in adults than in children, but is not confined to the latter.

Etiology.—The parasitic and syphilitic theories as to the origin of this affection are now discredited, and general opinion appears to incline towards a neurotrophic cause. That it occurs chiefly in debilitated subjects is certain.

Symptoms and course.—The disease begins as a minute red patch on the dorsum, or more rarely the edge of the tongue. As the spot increases in size it becomes shiny in the centre and acquires a yellowish, sharply-marked margin. There may be at first general erythema of the tongue, but this is rare, and, if present, transient. The rash slowly spreads out in all directions, like a ripple in a pond. After a while fresh patches form, and their intersection gives rise to the map-like appearance to which the name refers. Or rings may form within one another, each characterised by a bare, smooth centre and a well-defined pale edge. During the whole of its slow course the complaint may give rise to no symptoms; at

the worst there are itching of the patches and salivation. Occasionally the patches may be hyperalgesic.

The affection may last for years; it probably subsides spontaneously, though it has been said to lead to chronic ulceration.

Morbid anatomy.—The origin of the rash is not strictly superficial, but appears to depend upon inflammation in the derma. In the centre of the patches the horny layer is deficient. Opinions are still divided as to the involvement of the connective tissue.

Treatment is of little avail. Tonics should be given, and local astringent or emollient applications may alleviate discomfort, if present.

ii. *Xanthelasma.*—A few examples of this have been seen on the tongue in association with jaundice and xanthelasma of the skin.

iii. *Herpes.*—As previously stated, herpes zoster may attack the part of the tongue supplied by the lingual nerve. It is usually intensely painful and extremely intractable. The application of strong local antiseptics, under the influence of cocaine, appears to be of most service in causing the ulcers to heal. The general treatment of herpes, as a nerve disease, is of course indicated.

c. CHRONIC—i. *Leukoplakia.*—This very important affection is not limited to the tongue, which is, however, its chief seat. It is also found on the inner surfaces of the cheeks and lips, and occasionally on the gums and palate.

Etiology.—Leukoplakia is a disease of adult life, and is far commoner in men than in women. There must be some cause generally predisposing to its development, some personal defect which permits the specific action of the well-known factors in its causation, but as to the nature of this remote precursor we have no certain knowledge. Syphilis has, however, long been recognised as a predisposing, and excessive smoking as an exciting cause. Less important members of the former category are gout and rheumatism, and of the latter the consumption of strong spirits and hot liquids, and the irritation of carious teeth or ill-fitting tooth-plates (especially red vulcanite, Butlin).

Symptoms and course.—Leukoplakia may show extreme variation in course and extent, in one case appearing and persisting as one or two tiny "smoker's patches," in another covering the whole tongue with an indurated white coat. It may remain stationary or spread with greater or less rapidity, or after a period of apparent quiescence may break out into sudden virulence. When once established it seldom or never disappears; and its

white patches are an ever-present menace of epithelioma. A fairly typical case would have a history somewhat as follows: A man who has been exposed to one or more of the influences previously enumerated discovers a small patch on the dorsum of his tongue. His attention may be called to it accidentally, or he may be sensible of its slight tenderness; not infrequently it marks the spot whereon the mouthpiece of his pipe ordinarily rests. It is usually oval in shape, red in colour, glabrous and glossy in appearance; it may become coated with a yellowish crust, which peels off from time to time. If untreated, and more particularly if irritated, it slowly spreads over the whole tongue. This may now look as if covered by a thin white fur, but careful investigation proves that the coating is partly formed by and does not merely rest on the mucous membrane. Sometimes the colour is blue rather than white; sometimes the covering is thick and looks as if it were about to peel off. But if any desquamation occurs a raw and inflamed surface is exposed, and it is indeed the discomfort of this circumstance which may lead to the detection of leukoplakia; for the symptoms are usually trivial and almost always neglected. If the coating becomes thick a sense of dryness and harshness may be experienced, and if the patient has been warned as to the possible issue he may experience various forms of subjective discomfort, but he is not otherwise likely to be conscious of the existence of leukoplakia. Sometimes as the disease develops an enormous thickening of the characteristic covering supervenes, giving rise to the condition formerly termed ichthyosis linguæ. Finally, a large proportion of cases become epitheliomatous; a wart, a crack, or an ulcer appears, induration develops, and the fate of the tongue, if not of the patient, is sealed.

Morbid anatomy.—This may be described in general terms as that of a very chronic inflammation; there may or may not be precedent epithelial hypertrophy, but in any event there is after a while disappearance of the papillæ with formation of fibrous tissue beneath the epidermis. The epidermis may itself be thickened by increase in its horny layer. The passage into the condition of epithelioma is marked by the downgrowth of the epithelial cells and the formation of "nests."

Treatment.—There may be some hope of curing a "smoker's patch" and of staying the progress of an ordinary leukoplakia, but the ichthyotic condition is generally held to demand excision. The patient should always, if possible, be induced to discontinue smoking; the restricted use of tobacco may be grudgingly allowed in slight cases. Irritant foods are to be discountenanced, and substances

at extremes of temperature must not be put into the mouth. Local sources of irritation are, of course, to be removed. Antisyphilitic remedies are, as a rule, useless, as the disease is metasyphilitic, nor does the treatment of associated gout or rheumatism usually exert any favourable influence upon leukoplakia. Alkaline mouth-washes and antiseptic paints (*e.g.* 2 per cent chromic acid solution) may be useful in slight cases. Later on Butlin recommends ointments made up on a basis of lanoline. Caustics should never be employed. In the later stages any threatening patch should be freely excised.

ii. *The hairy tongue* (lingua nigra) — **Etiology.** — This rare and remarkable condition is the result of two factors; the filiform papillæ undergo elongatory hypertrophy and then become pigmented. The coloration has been attributed to the action of saprophytic moulds, one of which has, it is said, been cultivated as a black growth. Later observations, however, fail to support this view; in recent cases but few organisms have been found, and these have been normal to the mouth. It seems more probable that the colour results from the desiccation of the exposed epithelium. It is sometimes artificial. The essential cause of the phenomenon is unknown; it usually, but not invariably, occurs on the dorsum of the tongue in front of the circumvallate papillæ, a situation which is but little exposed to friction.

Symptoms and course.—So little discomfort is ordinarily caused by the condition that it is most often discovered by accident, as a small dark patch covered with what appear to be short hairs or bristles. These are black in the centre, light brown round the edges. They grow slowly, and after a few months fade away from the circumference first. The affection may recur or may persist unchanged for years. Sometimes the "hairs" grow very rapidly, and patients have been known habitually to shave them. Pain is very rare, and when present almost always slight.

Treatment.—Interference is not usually called for; shaving and scraping are followed by recurrence; a 2 per cent solution of salicylic acid may form a useful paint.

iii. *Local superficial glossitis.*—Circumscribed patches of inflammation are occasionally seen, chiefly at the edges and tips of the tongues of debilitated middle-aged women. They are painful and lead to anorexia, but do not ulcerate. The treatment is to relieve the general morbid condition, and to paint with a 5 per cent solution of chromic acid.

iv. *Sclerosing glossitis.*—This is a late manifestation of tertiary syphilis, and is characterised by a proliferative inflammation which

usually extends deeply into the organ. The surface is at first nodulated and may have a typical "hobnailed" appearance. After a while cicatrisation occurs and deep furrows are produced, leading to great deformity. In these furrows ulceration may from time to time develop, leading to distressing but temporary symptoms. In the earlier stages superficial plaques may be formed, which are hard, red, and painless, but may also ulcerate.

The treatment is to administer iodide of potassium and tonics. As a mouth-wash 1 in 1000 perchloride of mercury may be tried.

v. *Squamous glossitis.*—This rare affection, which is believed to be associated with dental caries, gives rise to the appearance of minute white spots on the tip and dorsum of the tongue. Removal of the white surface causes bleeding. The spots resemble aphthæ, but the course is chronic. The treatment is to touch with a pencil of silver nitrate.

ULCERATIVE GLOSSITIS

The diagnosis of an ulcer of the tongue may exceed in difficulty and importance any similar problem coming before the practitioner; the treatment of the more serious forms being mainly surgical, the diagnostic aspect of the question will be here chiefly considered.

The principal forms of lingual ulceration may be thus grouped:—

a. Simple—i. Acute; ii. Chronic.
b. Traumatic—i. From sudden injury; ii. From whooping-cough; iii. Dentitional; iv. From chronic irritation.
c. Mercurial.
d. Tuberculous and Lupous.
e. Syphilitic—i. Primary; ii. Secondary; iii. Tertiary, including gummatous.
f. Carcinomatous.

a. Simple lingual ulcers—i. *Acute.*—This is frequently, but not always, due to dyspepsia. It may, especially in ill-nourished children, be associated with buccal and lingual ulceration. The ulcers are multiple and chiefly situate on the tip and anterior part of the dorsum of the tongue. They are irregular in size and shape, red and irritable. They may also occur on the under surface of the organ. The fungiform papillæ usually appear to be large and red; the rest of the dorsum of the tongue is often furred. The condition yields readily to dieting and purgation; sometimes one or two of the more irritable ulcers may need touching with caustic.

ii. *Chronic.*—This form of ulceration sometimes supervenes upon

chronic superficial glossitis. It leads to the development of an intractable, callous ulcer on the dorsum of the tongue; there is as a rule little pain, but less evidence of healing. The treatment is that of leukoplakia; the last resource is excision of the ulcer.

b. Traumatic ulceration.—i. *From sudden injury*, such as incised or bitten wounds or scalding fluids; no special description is needed of this form of ulceration.

ii. *From whooping-cough.*—Little ulcers frequently arise on either side of the frænum as the result of friction by the lower incisors during the cough-paroxysms of pertussis. They are small and superficial, with a grayish, sloughy-looking floor. No treatment is required.

iii. *Dentitional.*—This is often seen on the under surface of the tongue of children at the time of the appearance of the lateral incisors. An oval ulcer forms on one or both sides, causes some little pain, and then fades away. If it be very troublesome the offending teeth may be varnished.

iv. *From chronic irritation.*—This is by far the most important variety of traumatic ulcer of the tongue. It is due in the vast majority of cases to the chafing of carious teeth or of an ill-fitting tooth-plate. It occurs most often in subjects who are out of health, and is always situate on the tip or edge of the tongue. The sore is at first minute, but as a rule rapidly grows, and is fully established within a week or so as a jagged, sloughy-looking ulcer surrounded by a zone of inflammation causing a good deal of swelling. After a time the acute symptoms and appearance subside, and considerable induration as a rule establishes itself in the base and edges. If neglected the ulcer may become epitheliomatous. Treatment is simple; the cause of irritation must be removed, the general health improved. Caustics should never be applied to such ulcers.

c. Mercurial ulceration.—This form of ulceration is only a part of general mercurial stomatitis in which, as already described, the gums first and chiefly suffer. Removal of the cause and the administration of a chlorate of potassium mouth-wash usually effect a rapid cure.

d. Tuberculous ulceration—i. *Lupous.*—This is almost unknown except as a complication of lupus of the face, especially when the nose is early or first attacked. It is a slowly-spreading ulceration associated with the development of the characteristic nodules and scar tissue. The treatment is that of lupus in general.

ii. *Tuberculous proper.*—The ordinary lingual tuberculous ulcer may be primary or, much more often, secondary to pulmonary or

laryngeal tuberculosis. It is commoner in men than in women, and in adults than in children. Its most typical site is at the tip of the tongue, but it may occur at the edge or on the dorsum (where it is often multiple) or frænum. It is at first a granular, irregular, indolent sore covered with a grayish slough, but after a while it becomes excruciatingly painful, in the writer's experience far more so than any other affection of the mouth save lingual papillitis. There is now profuse salivation, and the ulcer assumes a characteristic unhealthy appearance with a well-marked red edge, perhaps surrounded by a ring of yellowish secondary erosions, and a flabby, sloughy base, often extending down to the muscle. Induration is rare, but there is usually a good deal of œdema of the surrounding parts. The prognosis is of course gloomy; in the secondary cases the appearance of the ulcer is only too often a harbinger of the end.

Treatment.—Excision of the ulcer is the appropriate indication. This has led to quite favourable results in the case of primary ulcers and to considerable amelioration in secondary affections, when the patient's condition has permitted its performance. Failing this, palliative means are alone possible. The contiguous teeth should be fitted with a smooth plate or coated with copal varnish; irritating food should be avoided and the ulcer repeatedly dusted with a powder containing iodoform and cocaine. General treatment will naturally be requisite.

e. Syphilitic ulceration.—Ulcerations of the tongue in varying degree and extent, as well as of the mouth or palate, are frequently met with in *hereditary syphilis* (Vol. I. p. 378).

i. *Primary.*—This is rare; it usually appears as a superficial erosion with an indurated base; sometimes it involves the deeper tissues. The ulcer is of rapid growth, and the sub-maxillary and deep cervical glands are soon enlarged, often on both sides. There may be a moderate degree of pain and salivation. The treatment is general; the perchloride or, better, the bicyanide of mercury, may be used as a mouth-wash, 1 part in 10,000 for adults only, or 1 part in 1000 as a paint; or carbolic acid, 1 part in 100 as a wash, or 1 in 20 as a paint. Listerine diluted three or four times is a useful mouth-wash.

ii. *Secondary.*—The lingual manifestations of secondary syphilis are numerous and of frequent occurrence, developing within a few months after the disease is contracted.

Mucous patches.—These form white, well-defined plaques on the dorsum, warty condylomata on the under surface, and multiple ulcers surrounded by a red areola on the tip and edges. The ulcers are

usually shallow and but slightly painful. Other signs of syphilis are almost always evident. The treatment is the administration of mercury and the application of a 2 per cent solution of chromic acid to the patches.

Fissures.—At this stage fissures occur at the edges of the tongue where it is rubbed by the teeth. They are linear, not inflamed, but intensely sensitive. They often leave scars in which renewed ulceration may occur. The treatment is as for mucous patches.

iii. *Tertiary—gummatous.*—These ulcers may arise from the breaking down of a single large or numerous small gummata. In the former case they are always preceded by the appearance of a nodule, though the subjective evidence of this may be almost *nil;* in the latter they are of course multiple. The typical appearance is that of a large ulcer on the dorsum, or more rarely side, of the tongue, with everted edges and a yellow "wash-leather" slough covering its floor. The edge is ragged and undermined, and the surrounding epithelium forms thickened white patches often fissured. Other signs of tertiary syphilis are almost always present; the writer has twice seen gummatous ulcers of the tongue resulting from congenital syphilis, of which in one case there was no other evidence, though the history was conclusive. As a rule these ulcers disappear completely when the patient is placed upon a combination of iodide with tonics.

Fissures.—The tertiary fissures are long and deep, often leading to the most marked deformities. In them may form painful and very chronic ulcers. Astringent and antiseptic mouth-washes or ointments are required in addition to the ordinary treatment for tertiary syphilis.

f. Carcinomatous ulceration.—Cancer of the tongue may appear in many forms—as a fissure, a wart, a tiny nodule, an ulcer, and so forth. The importance of local irritation—such as excessive pipe smoking, carious broken teeth, or chronic syphilitic changes—in the determination of the disease cannot be over-estimated. The typical ulcer, when well developed, is at the side of the organ, and consists usually of a deep trench, in which the teeth play, with raised and indurated edges and a hard, almost stony base. There is a foul discharge, but at this stage often little pain, although severe shooting pains along various branches of the fifth nerve are frequently complained of. The glands near the angle of the jaw may be enlarged. Owing to spread of the growth into the structures forming the floor of the mouth, the tongue becomes fixed in position, and is moved with difficulty.

Among the very rare varieties of lingual ulceration is a very painful form that occurs under the tongue in old debilitated persons. The affection is often most intractable, but most benefit is likely to follow full doses of opium and quinine (Sheild).

A peculiar form of ulcer beneath the tongue of very exceptional occurrence is known as the "hæmorrhagic ulcer," from the constant bleeding that takes place in spite of all remedies. The ulcer is covered with flabby granulations of a deep livid blue colour, and slowly spreads. It is due to some form of acute sepsis, and is invariably fatal (Sheild, *Clinical Journal*, Sept. 24, 1902).

Pemphigus sometimes leads to the appearance on the tongue of the characteristic blebs, which, after forming thick crusts, leave intractable ulcers. Arsenic should be given, but the prognosis is very grave (see p. 70).

The diagnosis of lingual ulceration.—Briefly stated, the main fact which one wishes to ascertain with regard to an ulcer of the tongue is whether or not it is due to malignant disease. The following are among the principal practical considerations which may be of assistance in this connection.

a. History.—The age of the patient is of importance. Cancer may occur at almost any age, but is more likely the older the patient. It has been seen at and even before the age of thirty, and such early cases run a very rapid and severe course. The disease is much commoner in males. Traumatic ulceration is, however, also most frequent in later life.

Local irritation may determine ulceration in tuberculous or syphilitic patients, or may give rise to a simple sore in which cancer may or may not develop. Any inference from this condition must be made with caution.

The history of associated disease may be of great importance; the presence of advanced tuberculosis may convert a strong suspicion as to the nature of an ulcer into a certainty, but the strongest evidence of syphilis does not exclude the possibility of malignant disease, which may indeed develop in a syphilitic scar.

Theoretically speaking, the story of a rapid onset should go against cancer, but in practice one finds an almost incredible degree of ignorance and inattention, so that inoperable malignant disease may come under observation with a history extending only to a few weeks.

All severe lingual ulcerations lead to wasting, from the pain, the discharge, and the inability to take food. Sometimes, however, but not often, the cancerous cachexia develops to such a marked

extent as to yield some assistance in the diagnosis even of early cases. On the other hand, the general condition may appear good in cases of advanced cancer.

b. Site and characters.—Tuberculous ulcers are most often at the tip of the tongue, gummatous in the mid-dorsum, cancerous and traumatic at the side. But this rule is very far from being absolute. Cancer may develop in any part of the tongue, and, as a particularly baffling circumstance, in the scar of an old gumma. Induration is a marked characteristic of malignant disease, but is often found around traumatic ulcers and sometimes round those of tubercle and secondary syphilis. The stony feel of typical cancer is, however, not simulated by the other forms—with the exception, perhaps, of primary syphilis. Induration preceding ulceration is suggestive of gumma. White patches of thickened epithelium with fissures, and perhaps the scars of former ulcers, are strongly indicative of syphilitic origin.

Pain is not as a rule an early symptom of epithelioma of the tongue. By far the most painful of the common ulcers is the tuberculous. Cancer seldom, tubercle sometimes, gumma often produces multiple ulceration.

c. Histology.—This method of investigation is, if successful, pathognomonic of cancer. The epitheliomatous structure cannot be confounded with anything else. Histology is of much less use in the diagnosis of tuberculous ulcers, as giant-cells are rarely and tubercle bacilli almost never seen. The technique is simply to remove a shaving from the edge of the ulcer under cocaine.

d. Effects of treatment.—In doubtful diagnosis between cancerous and gummatous ulceration this method, though less positive than the last, is valuable. If gummata be treated by the internal administration of iodide, improvement is very rapid. This may, therefore, be legitimately tried for a week in a difficult case. It will not, of course, help if cancer have supervened in a brokendown gumma.

e. Glandular enlargement.—For practical purposes the diagnosis of cancer ought to be made before this appears. Early and rapid swelling of the glands is, however, a marked sign in primary syphilis. They are moderately swollen in secondary syphilis, and sometimes in tubercle, not in tertiary syphilis. They may be slightly swollen and very tender in traumatic ulcer, but this is always a gravely suspicious sign.

Non-inflammatory Affections, Chiefly of Congenital Origin

MACROGLOSSIA

This is a lymphangiomatous hypertrophy of the tongue, usually congenital, but occasionally consequent upon injury, such as division of the frænum or operations on the lower jaw. It is usually complicated by inflammation resulting from misguided interference. The essential change is a lymphangiomatous growth forming little blebs on the surface of the tongue; should the capillaries, which are dilated, rupture into them the vesicles assume a purple colour. Three changes may now supervene (Butlin and Spencer) which determine the future appearance of the swelling. Either the blood-vessels increase or dilate, or the lymphangiomatous tissue proliferates or inflammation is set up, leading to increase in fibrous tissue. Sometimes the condition remains unchanged from birth, but the rule is for it slowly to progress, sudden increases occurring from time to time as the result of attacks of acute glossitis. The tongue is often very sensitive, particularly in the early stages. Later on its surface is broken up into unsightly lobes and fissures, and is very liable to ulceration. The organ may attain an enormous size, protruding from the mouth and deforming the jaws. The treatment is by wedge-shaped excision.

A similar lymphangiomatous condition of the lips is known as *macrocheilia.*

Lingual hypermyotrophy is sometimes termed simple macroglossia. This condition is usually congenital and associated with defective intellect; it is especially common in cretins. The large lolling tongue is here the result of muscular hypertrophy; the patients are usually weakly and short-lived, and operative interference is seldom required.

CYSTS OF THE TONGUE

Dermoids are rare but interesting; they are usually situate in the midline between the geniohyoglossi and above the mylohyoid. The commonest places are immediately behind the incisors and immediately in front of the hyoid bone. They should be dissected out.

Hydatids are described, but are no more than curiosities.

NEW GROWTHS OF THE TONGUE

These do not concern the physician unless they cause ulceration.

NERVOUS AFFECTIONS OF THE MOUTH

Motor.—General *spasm* of the tongue and lips is well known in chorea and other nervous diseases. It may also occur as a form of or factor in stammering. Involuntary protrusion of the organ from tonic spasm of the geniohyoglossus is also described. Where organic disease is present it must, of course, be alleviated, but most of these conditions, as well as of those about to be described, belong to the category termed "functional," and call for the general treatment of nervous instability (see Vol. III.). Spasm of the muscles of mastication, which are supplied by the fifth cranial nerve, is far more often tonic than clonic. In the former case the condition, when extreme, is familiar under the name of "lockjaw." It may be the first symptom of tetanus, or the most persistent of tetany; it is at times hysterical, at others the result of peripheral irritation, usually in the region of distribution of the trigeminal; or it may arise from organic pontine disease irritating the motor nucleus of that nerve. Clonic spasm is usually rapid, producing "chattering of the teeth," and merely a part of general clonic spasm, or associated with paralysis agitans. It has, however, been recorded as an isolated phenomenon, chiefly in middle-aged women. Much more rarely the clonus takes the form of "occasional, severe, single contractions" (Gowers). These may be seen sometimes in chorea, very infrequently without other nervous affection. The tongue and cheeks are liable to be bitten.

Treatment is that of the underlying disease, if any; failing this, nerve tonics, counter-irritation, and galvanism are indicated.

Tremor of the tongue and lips is an early sign of general paralysis of the insane. It may also occur in chronic alcoholic and mercurial poisoning and in paralysis agitans.

Paralysis of the tongue, lips, or masticatory muscles may be seen in various lesions of the brain and cranial nerves, and may by its characters and distribution be of value in diagnosis. When due to disease of the lower motor neurone (as in, *e.g.*, bulbar palsy) it is attended by wasting. Inability to protrude the tongue is an early sign of that mysterious and fatal disease, myasthenia gravis. Paralysis of the external pterygoid, which is supplied by the motor division of the trigeminal nerve, renders the patient unable to move

the jaw towards the unaffected side. When the mouth is opened the chin deviates towards the paralysed side.

SENSORY—*a. General sensation.—Anæsthesia* of the buccal mucous membrane may result from central or peripheral nerve lesions, more frequently the latter. Hemianæsthesia is much commoner and is usually a part of general hysterical hemianæsthesia. All forms of common sensation are equally affected, and "trophic" ulcers may appear on the diseased side.

Hyperæsthesia and *paræsthesiæ*, especially tingling and burning sensations within the cheeks or at the tip of the tongue, are not uncommon, particularly in women at puberty and the climacteric. They are often seen in the insane. The paræsthesiæ may be followed by anæsthesia or by buccal neuralgia.

Buccal neuralgia may attack an entire moiety of the buccal mucosa or be limited to half the tongue. In the first case it comes on in spasms, like those of convulsive tic, at repeated intervals. Mastication and speech are difficult and there is profuse salivation. The lingual form (glossodynia) is usually of a more continuous type and is frequently preceded by paræsthesiæ. There are tender spots. The pain often disappears during a meal and returns later, but the reverse may more rarely be the case. The complaint, which is as a rule very intractable, is essentially a variety of trigeminal neuralgia and owns the same general and local causes. Its subjects are usually neurotic. The treatment is accordingly general; locally sedative rather than irritant applications should be employed. Division of the lingual nerve or its branches has been recommended in extreme cases.

b. Taste.—It will be, of course, remembered that the sense of taste proper has to do with the appreciation of those qualities alone to which the names sweet, salt, acid and bitter have been applied; the recognition of all other flavours is effected through the olfactory nerve.

Ageustia, or loss of the sense of taste, may be temporary, when it is often caused by dryness of the mouth or its irritation by spices, or permanent, when it connotes some affection of the chorda tympani, lingual or glossopharyngeal nerve, or possibly of the deeper parts of the brain. Ageustia is often a sign of functional disease; it may then be extremely local. Treatment is as a rule of but little avail, though the use of the constant current has been asserted to be of use.

Hypergeustia is an occasional hysterical symptom.

Parageustia may occur in gastric troubles, and is sometimes

responsible for the dietary whims of pregnant women. It is frequently seen in lunatics, in whom, however, it is with difficulty distinguished from gustatory hallucinations.

Reflex neuroses of the mouth, as described by Baumgarten, Engelhardt, and others, are very rare, and of little clinical importance.

AFFECTIONS OF THE SALIVARY GLANDS

Ptyalism or Salivation

This is but seldom an "idiopathic" affection, though it may occur without assignable cause in sucking infants. Care must be taken not to confuse the dribbling of saliva from a constantly open mouth with true super-secretion. In the latter condition the specific gravity of the saliva always falls and may be as low as 1001; the amount secreted in a day is said to have reached 25 pints. Occasionally the specific gravity is at first increased, in one case to 1059.

The most frequent cause of salivation was formerly the abuse of mercury; other toxic agents which may produce it are pilocarpine, potassium iodide, and arsenic. In all these cases personal idiosyncrasy has a great deal to do with the ease and rapidity with which salivation is evoked. Salivation may also accompany acute infections, such as hydrophobia and variola; in the latter case and in some others it may further be associated with the commencement of defervescence ("critical ptyalism," Kraus). Another class of causes comprises certain general conditions, such as pregnancy and lactation, and also occasionally affections of the nose, stomach, and intestine. Finally, ptyalism may arise from purely nervous causes, acting in the main either directly or indirectly upon the chorda tympani. Thus it is an almost constant complication of tic douloureux; in bulbar palsy the element of inability to swallow is added. The enumeration of the causes suggests the various modes of appropriate treatment, the salivation being almost always a symptom and not a disease. If, however, direct treatment is necessary or desirable, atropine may be given, or preferably opium or bromide. Potassium chlorate is often of great value.

Xerostomia

This condition is the reverse of the last. It may occur temporarily, as from fear, or be habitual, as the result of diabetes or of

chronic mental distress. The deficiency of salivary secretion in fever has already been noticed. Xerostomia is sometimes congenital, but most frequently occurs in women over middle age; when it is marked the sense of taste is much interfered with. The tongue is red, denuded, and cracked, and like the inner surface of the mouth generally is very dry, the mucous membrane being pale and shiny. The parotid glands are sometimes enlarged. And in a few cases the nasal and ocular mucous membranes and even the skin have been similarly affected. The **treatment** of chronic cases is disheartening. Pilocarpine is the best drug, but causes inconvenient sweating; jaborandi extract is generally useless as containing an inappreciable amount of alkaloid. The condition can be alleviated by repeatedly smearing the tongue with simple ointments.

Salivary Calculi

Calculi in the ducts of the salivary glands—especially the submaxillary—are not uncommon. They have been known to follow trauma, acute inflammation, and the administration of mercury. They are usually solitary, but occasionally occur in the form of multiple rice-like grains in the secondary ducts. In most cases they consist of a microbic network—the leptothrix buccalis being especially abundant—in and around which lime salts have been deposited. The presence of a foreign body as a nucleus is an event of the extremest rarity. Sialolithiasis is commonest in middle age; the largest recorded stone weighed more than three ounces. The early symptoms are very slight, but after a while the duct is obstructed and the gland swells. Even then not much discomfort may be felt, except in the case of stones in the submaxillary duct, when there may be severe pain on eating, and particularly on swallowing. If the condition is not attended to suppuration may occur round the stone, which may be eventually buried in a dense mass of fibrous tissue, into which fistulous tracts open. The diagnosis is easy unless the stone is embedded in an inflamed gland, when a new growth may be simulated. The treatment is to open the duct from within the mouth and extract the stone.

Ranula

This name is applied to a retention cyst of a salivary or mucous gland. The obstruction is probably due in most cases either to a

congenital malformation, or to a special inflammation of the gland duct (especially the kind termed by the Germans sialodochitis fibrinosa). Ordinary inflammatory affections of the mouth do not appear to lead to ranula, but it may be caused by the pressure of a tumour on the duct. The commonest form occurs in the sublingual gland, giving rise to a tense, fluctuating swelling of a purplish colour beneath the tongue; its pressure causes atrophy of the rest of the sublingual gland. Dilated veins are seen over the surface of the cyst; there is, at first at any rate, no pain. Blaudin's gland, the incisive glands, and the submaxillary salivary glands may be similarly affected. Occasionally there is an acute development, as from sudden obstruction. In such cases there is great pain and discomfort.

The treatment is to excise a portion of the cyst wall, and to rub the remainder with silver nitrate.

Inflammations of the Salivary Glands

Mumps has already been described in Vol. I. p. 337.

Acute secondary metastatic parotitis.—This is a secondary parotitis with a marked tendency to suppuration. The researches of Hanan, Dittrich, and others show that where specific micro-organisms are present they infect the gland through its duct, and not by the blood stream. The term "metastatic" should be accordingly abandoned. The chief diseases which this form of parotitis complicates are typhoid, puerperal sepsis, typhus, dysentery, and intermittent fever. It may appear in pyæmia from any cause, but particularly of abdominal origin. It is described as occurring also in measles, scarlatina, pneumonia, and small-pox, and has even been seen to follow the continued use of nutrient enemata. As a rule, it is an event of the gravest significance, particularly if appearing in the early stage of a disease. The inflammation is limited to the parotid gland, often to one alone, but the abscess may burst into deeper structures.

The treatment, apart from that of the primary cause, is by belladonna fomentations, followed by free incision when pus appears.

Tubercle and syphilis may attack the salivary glands, more especially the parotid, but so rarely as to be of little importance.

New growths are common in the parotid, and are usually of a mixed type, often containing cartilage.

Bertram Abrahams.

DISEASES OF THE PHARYNX

VASCULAR LESIONS OF THE PHARYNX

Anæmia.—This is not an idiopathic affection, but a complication of grave forms of general anæmia, especially pernicious anæmia and that following severe and repeated hæmorrhage. It is of interest chiefly as leading to marked atrophy of the mucous membrane, which may become so thin that the muscular fibres can be seen through it.

Hyperæmia.—This is extremely common; it accompanies all inflammations in the neighbourhood, and may also occur independently, from reflex vasomotor causes. An acute hyperæmia of the latter type may be seen in digestive disorders, at the menstrual period, and in association with gout. There is usually an uncomfortably excessive secretion of mucus. Passive hyperæmia occurs in association with cardiac disease, with mediastinal tumours, and with other affections interfering with the return of blood.

Œdema.—Apart from œdema associated with general anasarca or with local inflammation, at least two forms of acute dropsy of the pharynx are known. The one is urticarial in nature, and has been described in connection with urticaria of the skin after a meal of shell-fish. The other is of the type known as angioneurotic. It is induced in "nervous" subjects by a chill, or comes on in them without assignable cause. Within twenty-four hours the pharynx may be so swollen as to threaten suffocation. The grave infective disorder known as pharyngeal phlegmon may be suspected, but there is no fever and the swelling disappears as rapidly as it arose.

Pharyngeal Hæmorrhage. — This is seldom of sufficient severity to entail danger; its chief importance is derived from the alarm which it may cause by simulating hæmoptysis. It may be the result of the various conditions (scurvy, hæmophilia, leukæmia, etc.) which are conveniently termed hæmorrhagic diatheses; of certain acute infective processes, such as variola and typhus, or of chronic diseases like nephritis or diabetes; it may connote an epithelial lesion occasioned by acute or chronic ulceration or injury by a foreign body; or it may be due to the bursting of veins—especially at the root of the tongue and at the back of the tonsil—which either are varicose or have become dilated. Such hæmorrhages are not infrequent in drunkards, in

whom portal obstruction may be combined with some chronic pharyngitis. Hæmorrhage from the naso-pharynx is not infrequent in connection with adenoid vegetations; the pillows of children suffering from these are often soiled by blood-stained saliva. It may occur in the atrophic rhinopharyngitis of adults. If the blood trickles into the respiratory passages during sleep it may be hawked up in the morning mixed with frothy mucus. Careful examination of the pharynx should establish the diagnosis, but the chest ought to be thoroughly overhauled in every case.

The **treatment** must aim at combating the cause. The depletion of the portal circulation, the administration of calcium chloride in hæmorrhagic diatheses, and so forth, are obvious remedies. Locally the galvano-cautery may be applied to the bleeding spot; where there is more general oozing adrenalin is a valuable styptic. In severe bleeding from an inaccessible situation the common carotid may have to be tied.

ACUTE INFLAMMATIONS OF THE PHARYNX

ACUTE CATARRHAL PHARYNGITIS.—This is a very common affection. It may occur alone or as a symptom or complication of other complaints. It is best considered under the following heads:—

SIMPLE.—This is often attributed to *cold*, which is no doubt an exciting cause, though less frequently than is generally assumed. It is very hard in such cases to exclude microbial influence. *Trauma*, as by scalding fluids or irritating gases, is also a factor in a certain proportion of cases.

SEPTIC.—"Drain throat" and "hospital throat" are familiar examples of this. The excitants are probably pus cocci, and the affection is quite common among those engaged in nursing septic cases. This form of sore throat is much predisposed to by the habit of mouth-breathing, and hence particularly affects the subjects of nasal obstruction. The writer has noticed that the inhalation of the filthy dust from the surface of wooden roadways leads to sore throats in mouth-breathers, but to "hay-fever" in nose-breathers. That air in its passage through the nose is strained of most of its germs is a familiar fact.

SYMPTOMATIC.—Sore throat is an early symptom of many exanthemata, *e.g.* scarlet fever, measles, and roseola, and is also frequently seen at the onset of an attack of acute rheumatism. It is, moreover, a sign of certain drug intoxications, such as

antimony, belladonna, and potassium iodide; there is evidence, too, that it is sometimes associated with gout.

SECONDARY.—Inflammations of neighbouring parts readily spread to the pharynx, which may hence be secondarily involved in tonsillitis, various forms of stomatitis, and catarrhal rhinitis. That catarrhal pharyngitis may complicate coryza is a matter of everyday experience. It must not be forgotten, also, that the course of chronic pharyngitis is liable to be complicated by acute attacks.

Symptoms.—These are often trivial, yielding readily to domestic remedies. The throat feels sore, especially during the swallowing of solids, and there is discomfort and stiffness in speaking. A dry, tickling sensation is usually present, and the patient is led to hawk and cough, thereby increasing the malady. After a day or so a small quantity of sticky muco-pus is secreted and the dryness accordingly lessened. There are sometimes malaise and pain in the back, but it must not be forgotten that cases in which these are present are frequently influenzal. Upon examination the pharynx is at first seen to be red and dry; later on some secretion is effused upon it. The process usually spreads to the nose and often to the trachea; the tonsils and mouth are always hyperæmic and the former swollen.

Treatment.—A mild case will call for nothing beyond a hot bath, a dose of Dover's powder, a calomel purge, and perhaps some guaiacum or rhatany lozenges. The primary cause of symptomatic cases must of course be remedied. In severer attacks a menthol spray may be given and a cold compress applied to the throat; the patient had better remain in bed for a day or two. Inhalations of vapor benzoini are useful if the larynx be involved. Stoerk recommends vigorous painting with strong silver nitrate solutions in severe cases; care must be taken to keep the remedy out of the larynx.

ACUTE SEPTIC PHARYNGITIS.—Semon has brought forward valuable evidence in support of the view that the different septic inflammations of the pharynx, larynx, and neck are due to varying degrees of virulence in the same pathological process. It will be, accordingly, convenient to study the acute cellulitis of the neck, known as Ludwig's angina, or angina Ludovici, in connection with acute septic pharyngitis. The courses they run are clinically similar, and each may be produced, it seems, by either strepto- or staphylococci; both are closely allied pathogenetically to erysipelas. The cause, then, of these conditions is infection with septic micro-organisms; their histories will depend upon the virulence of these agents, the resisting power of the individual, and the site of invasion.

Symptoms.—The onset is usually marked by a rigor, with a sharp rise of temperature; there may or may not have been a preliminary period of malaise. Acute pain in the throat is an early symptom, and there is as a rule great difficulty in swallowing. There is at this stage intense hyperæmia of the pharynx and tonsils. The affection may cease at this stage, or after the occurrence of a little local necrosis; more often it extends and becomes œdematous. The extension may be to the larynx or the cellular tissue of the neck, or there may be simply a more intense and severe involvement of the pharynx. If the larynx be implicated urgent dyspnœa will, as a rule, call for tracheotomy; this complication is usually fatal. When the inflammation remains restricted to the pharynx there is much swelling of the mucosa, which assumes an angry purple tint, and is often flecked by submucous hæmorrhages. Rapid suppuration may follow (Senator's "acute infectious phlegmon"), burrowing in all directions and implicating the deeper structures of the neck. Lastly, when the process extends early to the cellular planes of the neck the condition known as Ludwig's angina is set up. There is brawny induration of the connective tissue, which does not pit on pressure, spreads uniformly, and has a well-defined border, a "rounded edge" so to speak. A similar swelling is found under the tongue and along the inner edge of the mandibular ramus; the glands are not involved, whence and from the rapidity of its course it may be inferred that the infection spreads along the veins. There may be high fever from the beginning, but the worst cases often run a subfebrile course, with delirium and eventually coma. Albumin and sugar are sometimes found in the urine. The prognosis is always grave. If recovery occurs in the phlegmonous forms it is usually after much sloughing. Death may follow from septic intoxication (in these cases there is often but little suppuration or fever), from asphyxia by laryngeal involvement, from septicæmia or exhaustion following gangrene of the parts involved, or from septic pneumonia resulting from inhalation of septic particles. Occasionally the erosion of a large vessel leads to fatal hæmorrhage.

Treatment.—This must be vigorously carried out. Large doses of tinct. ferri perchlor. and of quinine must be given, and stimulants should be freely administered. Antistreptococcic serum may sometimes be of use. Preparations for intubation or tracheotomy should be at once made, as urgent dyspnœa may supervene at any moment. Digitalis should be given if the pulse shows signs of failure. Locally little can be done beyond applying

ice to the neck externally, giving the patient some to suck, and spraying the pharynx with cocaine. Obvious and accessible foci of suppuration should be freely evacuated.

Membranous Pharyngitis.—Simple pharyngitis is seldom so intense as to cause the outpouring of a fibrinous exudate. This, however, has upon several occasions been observed as a complication of influenza (Stoerk, v. Réthi). The cases in which it occurs are not those in which the throat is very painful; they are characterised by a high, but very evanescent fever. The membrane begins to clear off and the temperature falls within two or three days. The course renders confusion with diphtheria almost impossible. No bacilli are, it need hardly be said, to be found in the exudate. The tendency being towards rapid and spontaneous cure, no treatment other than warm gargles is called for.

Gouty affections of the pharynx.—"Gout of the throat" is a diagnosis which is frequently made, it is to be feared, on insufficient grounds; it is nevertheless established that certain pharyngeal affections are undoubtedly of gouty origin. They may be acute or chronic.

With regard to the acute form, all that need be said is that exposure to cold is liable to induce attacks of pharyngitis or tonsillitis in gouty subjects, which are particularly characterised by severe pain and spasm, quite out of proportion as a rule to the objective signs. The inflammation is usually patchy, and is prone to attack particularly the lateral walls of the pharynx. These are also especially affected and usually thickened in chronic gouty pharyngitis. There is here again abnormal pain and sensitiveness in the throat, and the inflammatory changes are of a patchy nature. Tophi have been described as forming under the mucous membrane.

The local treatment does not differ from that of pharyngitis or tonsillitis due to other causes; the general is that of gout.

ACUTE DISEASES OF THE TONSILS

The functions of the tonsils are still obscure. Their lymphoid structure and situation at the portal of the respiratory and alimentary passages seem indicative of service in the protection of those passages against infective invasion. The fact that pneumococci, tubercle bacilli, pus cocci, and other hostile organisms are frequently found in them is alleged by many to be evidence that the tonsils act as a common source of serious infection. There is

no doubt that repeated attacks of inflammation deprive the tonsils of their resisting power, and render them foci of disease; but it is most probable that the healthy organs form part of a protective and not of an offensive mechanism. The need of protection doubtless diminishes with age for the tonsils normally atrophy in early adult life.

Acute Tonsillitis.—Acute inflammation of the tonsils is commonest in young people. It sometimes occurs in epidemics; in this case it may spread by contagion from person to person, but is more probably due to some hygienic defect, such as neglected drainage. Micro-organisms are always present in acute tonsillitis, and it is consequently impossible to prove the existence of cases due to other than microbic causes. The chief pathogenic organisms found are staphylococci and streptococci of various kinds, the pneumococcus, the micrococcus tetragonus, and the diphtheria bacillus. Should the last named be present in a swab and prove virulent on injection into guinea-pigs, the case must be treated as one of diphtheria, no matter what the clinical phenomena may be. It is said that the complaint runs a milder and more circumscribed course when due to staphylococci than to streptococci. An attack of acute tonsillitis predisposes to others, and is likely also to lead to some tonsillar hypertrophy; hence it is that most of the patients one sees suffering from it have enlarged tonsils from the outset. Two chief forms of acute tonsillitis are recognised, the lacunar and the parenchymatous; the former appears to be the commoner in children, and more apt to occur in epidemics; the latter in young adults, though a case has been recorded at the age of seven months (Hall). Recurrences are usually, but not invariably, of the same type as the original attack.

Lacunar or Follicular Tonsillitis—**Symptoms.**—These almost always set in with considerable acuteness. There is chilliness—sometimes even to the extent of a rigor—with headache, malaise, and the other indications of fever. The temperature is high, often reaching, or even passing, 104°. The voice is thickened, muffled, or guttural. The throat feels sore, and swallowing is painful. The appetite is lost, but thirst is complained of. The tongue is thickly furred, the bowels are confined, and the urine is febrile and sometimes albuminous. There may be slight swelling of the glands at the angle of the jaw and of the submaxillary. There is not infrequently a general cutaneous erythema, patchy or diffuse; cardiac murmurs are often to be heard, either as the result of acute dilatation or of co-existing valvular disease. Extension of

the inflammation may lead to deafness. Although in acute tonsillitis there is considerable blocking of the throat there is rarely any difficulty in breathing.

It is now well established that a considerable number of attacks of acute tonsillitis—both lacunar and parenchymatous—are followed within a fortnight or so by acute rheumatism. The evidence very strongly suggests that the organisms which one must believe are the cause of acute rheumatism effect an entrance in these cases by way of the tonsils, which offer a stalwart resistance, as shown by the reactive inflammation. The liability for this resistance to be overcome by the microbes is much enhanced if the tonsils have been damaged by previous inflammatory attacks. The writer has discussed the relations of the tonsils to rheumatism at length in a paper in the *Transactions of the Clinical Society of London for* 1899. (See also Vol. I. p. 301.)

The patient is much pulled down and may appear to be very ill, out of all proportion to the extent of the local affection, but under appropriate treatment the complaint abates in three to seven days. By the methods to be mentioned great relief is obtained in twenty-four hours in almost every case.

Morbid appearances.—The tonsils are at first slightly swollen and rather red. After a day or two the swelling increases, though never to an extreme degree, and the redness deepens. Yellowish white spots now begin to appear upon the surface, and may extend and fuse together so as to form a "pseudo-membrane," which is, however, easily broken down and detached without leaving a raw surface. The exudation consists of epithelial cells, lymph corpuscles, and detritus, mixed with micro-organisms; it first appears in the crypts or lacunæ of the tonsil, whence the name applied to this form of inflammation.

Treatment.—In the writer's experience nothing acts so well as perchloride of iron. The formula for an adult is

℞ Tincturæ ferri perchloridi, ♏x.
Potassii chloratis, gr. v.
Glycerini, ℥j.
Aquam ad ℥j.

to be gargled first and then swallowed every four hours. Children who are too young to gargle should simply swallow an appropriate quantity of the remedy, which may be regarded as almost specific. Salicylates are of value in overcoming rheumatic symptoms when present, and of course speedily lower the temperature; but it is

doubtful if they exert much influence upon the throat condition. Aconite is less useful here than in the parenchymatous form. Painting with guaiacol (50 per cent), cresol (2 per cent), protargol or tinct. iodi, is recommended by various authorities. The most useful is a 10 per cent cocaine solution, and that merely for the purpose of allaying pain, when this is so severe as to prevent the patient from swallowing.

It need hardly be said that during the febrile stage the patient must rest in bed and live on fluids. The bowels must be kept freely open. Cardiac complications should be sought, and if found treated. After the attack there is much weakness and lassitude; quinine, iron, and change of air are indicated. The patient's sanitary surroundings must be investigated; if defective their neglect will infallibly entail recurrences.

Parenchymatous tonsillitis—**Symptoms.**—In this form—popularly known as *quinsy*—there is much swelling and a tendency to suppuration, either in the body of the tonsil or more frequently in the supratonsillar fossa (so-called "peritonsillar" abscess). One or both tonsils may be affected, the one after the other, or occasionally both together, but as a rule only one suppurates. The local symptoms are very severe: intense pain in swallowing, swelling, and stiffness at the angle of the jaw, sometimes even inability to open the mouth. There is fever, but to a less extent than in the lacunar form. Convulsions have been recorded as a rare complication. The tonsils are seen to be deep red or purplish, much swollen, almost or quite meeting in the middle line; and the uvula and soft palate are œdematous. After a while a boggy protrusion appears, usually on the upper portion. This indicates suppuration, and unless an incision be made will be the place where the abscess bursts, which is the natural termination of the malady; but it may resolve without going on to suppuration. The abscess has been known to extend into the neck or even the chest, and is recorded to have burst into the carotid artery. It may be complicated by œdema of the glottis, and so prove fatal. Gangrene is another possible sequence. Orchitis and ovaritis have been noted.

Treatment.—Before suppuration is evident the best remedy is aconite. An adult should have three minims of the tincture every two hours, a child one minim. A brisk purge should also be given. Some authors prefer large doses of sodium salicylate to aconite; or it may be well to combine these remedies. Both these drugs must be given with caution if signs of organic heart disease are present. Applications every few minutes of bicarbonate of soda,

as powder, have been recommended, or insufflations of sulphur. Sucking ice gives considerable relief. As soon as pus can be reasonably suspected it must be evacuated, best by a linear incision through the junction of tonsil and palate above. One incision, if properly made, will suffice, except in the so-called "multilocular" abscesses, where a succession of pockets will have to be opened. After the operation the temperature quickly falls, and great relief follows; this is enhanced by the use of warm gargles and guaiacum lozenges. The general health will require attention.

Ulcerative tonsillitis.—This term was formerly applied—and still attaches in popular parlance—to the lacunar form. But an acute ulcerative tonsillitis is, though rarely, met with in debilitated and anæmic subjects. The ulcers may be as much as one-third of an inch in diameter. They are of an indolent type, covered by a grayish slough, and not surrounded by any zone of inflammation. There are few general symptoms beyond those of the predisposing condition. Locally the throat feels sore, but as the tonsils are little if at all enlarged, there is no great difficulty in deglutition. The treatment is general, tonics and fresh air; locally, applications of tannin or perchloride of iron do most good.

Throat Complications of Acute Infectious Diseases

Scarlet fever.—The condition of the throat in this disease has already been described in Vol. I., and will be again referred to when the differential diagnosis of acute faucial inflammations is considered. One or two points may, however, be here noted. A membrane may appear in the throat of a scarlatinal patient either early or late in the disease. In the first case it is usually a "pseudo-membrane," consisting of pus cells and débris; in the second it may be diphtherial. Diphtheria is almost always a late complication of scarlet fever. Secondly, it is stated that in patients who have suffered from scarlet fever, and have again been exposed to it, a sore throat may develop, which, though unaccompanied by rash, having the appearance of a simple or sloughing tonsillitis, may communicate the virus of the disease.

Measles.—Punctate red spots may be seen on the palate before the exanthem appears. There is always some pharyngitis at an early stage of the disease.

Rötheln.—Sore throat with congestion of the pharynx is a constant initial symptom.

Varicella.—The vesicles are often seen on the pharynx and tonsils; they form little ulcers, which heal after a few days.

Rabies.—The pharynx is intensely red—more than in any other disease (Stoerk)—and any attempt to swallow leads to painful reflex spasm.

Small-pox.—Pocks may be seen on the pharyngeal mucous membrane, or in malignant cases this may be studded with hæmorrhages. The laryngeal complications are much more dangerous than the pharyngeal, which they usually mask.

Influenza.—The throat is very frequently affected, the onset of the disease being often ushered in by pharyngitis or lacunar tonsillitis. Sometimes suppuration occurs in the tonsil; sometimes a false membrane is formed. A vesicular eruption on the soft palate is also described in influenza. Influenzal throat affections often fail to clear up completely, and a condition of chronic tonsillitis or of pharyngeal catarrh is apt to persist.

Typhoid fever.—The initial sore throat is slight and merely erythematous. There are sometimes a few small superficial ulcers of the fauces and soft palate. True diphtheria has sometimes been seen to complicate typhoid.

Acute rheumatism.—In the paper already referred to, the writer deduced from the study of a large number of cases that the affections of the throat associated with acute rheumatism are mainly of two kinds, faucial erythema and acute tonsillitis; the latter may be either lacunar or parenchymatous. Faucial erythema is an initial symptom, and appears when the temperature first rises; it is present in at least 50 per cent of adult cases; it is apparently less frequent in children, though the slightness or absence of joint symptoms in them may lead to its not being early enough sought. Acute tonsillitis, on the other hand, precedes the rheumatic attack by, as a rule, two to three weeks. Dr. Watson Williams states that most cases of acute follicular tonsillitis are rheumatic; my own statistics show a personal or family history of rheumatism in about 54 per cent. I find that about 25 per cent of attacks of rheumatism in adults are preceded within three weeks by tonsillitis, that there is a throat history in more than 50 per cent of children affected with chorea, and that two children out of every three who develop heart disease without having had chorea have suffered from sore throats. The tonsillar affection is identical in rheumatic and non-rheumatic forms, and the action of salicylates affords, in my experience, no differential criterion.

The Differential Diagnosis of Acute Sore Throat

Correctly to diagnose an acute faucial inflammation is a matter of grave importance; it may be one of the simplest problems in clinical medicine or one of the most difficult. The three most dangerous forms of acute sore throat are the diphtherial, the scarlatinal, and the acute septic. The last-named, unfortunately, begins in a manner which is not, in the present state of our knowledge, to be distinguished from the onset of simple pharyngitis; and it must not be forgotten that diphtherial infection may, so to speak, be grafted on a throat attacked by lacunar tonsillitis, measles, or scarlatina. In the last-mentioned case, however, diphtheria does not as a rule supervene till the patient is beginning to convalesce from the scarlet fever. The other common acute inflammations of the throat which may lead to diagnostic difficulty are tonsillitis, especially lacunar, those of other acute specifics, and that of secondary syphilis. Too much stress cannot be laid upon the fact that an error in diagnosis may entail the gravest consequences to the patient, to those in contact with him, and to the reputation of his medical attendant; in a doubtful case, therefore, no aid to a correct opinion should be neglected. The principal guides are—

1. The history of and circumstances surrounding the patient.
2. The physical examination of the throat and its neighbourhood.
3. Associated symptoms and signs, general and local.

History and surrounding circumstances.—The presence of an epidemic is a very important guide; it is particularly helpful if one remembers that in an epidemic there is mingled with the typical cases, which are of course in the great majority, a certain proportion in which the symptoms and physical signs deviate from those normal in the disease. A knowledge of this fact will often lead to the suspicion of diphtheria, scarlet fever, or lacunar tonsillitis, as the case may be, when the patient's condition is anomalous; further investigation in accordance with the clue may then result in the satisfactory detection of the disease. *Per contra* the knowledge of an epidemic of measles or rötheln, though it should not lessen caution, may allay anxiety. The patient's history may also be of great assistance. Thus a previous attack of scarlet fever ordinarily excludes a recurrence, though a throat condition (see p. 110) may arise, which is said to be infectious. Previous

infection with diphtheria, on the other hand, tends to predispose rather than to protect. A history of rheumatism may be suggestive of lacunar tonsillitis, particularly if the patient's throat is frequently affected; it will not, of course, exclude the supervention of another acute specific. A history of recent syphilis will, of course, be a valuable indication. Hospitalism, if there is definitely known to have been no exposure to diphtheria or scarlet fever, will make for lacunar tonsillitis. The influenzal sore throat is almost always a lacunar tonsillitis.

The physical examination of the throat and its neighbourhood.— The chief acute affections which may resemble each other in appearance are diphtheria, scarlet fever, and lacunar tonsillitis, and it is to the diagnosis of these that attention will be directed. To begin with, it is worthy of remembrance that the throat is, as a rule, much easier to examine in diphtheria than in either of the other conditions. In tonsillitis, on the other hand, pain is often a pronounced symptom, and there may be acute dysphagia. There is difficulty in opening the mouth, and in children there may be great opposition to the inspection of the throat. The condition in scarlet fever is, as a rule, similar, but less severe.

In the fauces the first thing to look for is membrane. Its absence will, of course, tend to allay the suspicion of diphtheria, but it must never be forgotten that in a certain number of cases of this disease no membrane is seen. These are by far the hardest to diagnose, and in them there is hence a severe risk of post-diphtherial palsy. Physical examination is of little use here, except that scantiness of secretion is suggestive of diphtheria, lacunar tonsillitis leading usually to a copious stringy, sticky exudation. The associated signs, particularly those connected with the temperature, the urine, and the knee-jerks, must be most carefully investigated; in this class of case, too, bacteriology may render invaluable service. To distinguish between scarlatina and acute tonsillitis by the throat alone may be difficult. The former is always bilateral and of approximately equal extent on the two sides; the latter may be unilateral, and is almost always much more marked on one side than the other.

The presence of small yellow spots scattered over the tonsils is sure evidence of lacunar inflammation; but without the aid of a bacteriological examination it cannot be held to exclude diphtheria. Symmetrical, shallow, irregular ulcers on the tonsils similarly indicate secondary syphilis. The punctate "endanthem" of measles is, if once seen, easy to recognise. The vesicles of varicella and

papules of small-pox appear concomitantly with the skin manifestations of those diseases.

Should a membrane be present, attention must be directed to its character and extent. In diphtheria the exudation is closely attached to the mucous membrane, and is hence peeled off with difficulty, leaving a raw, bleeding surface. It almost always extends to the uvula and soft palate, across which it can be seen to creep from day to day. In lacunar tonsillitis the membrane is a "false" one, *i.e.* it does not contain fibrin, and is merely a caseous exudate containing broken-down epithelial cells and leucocytes with micro-organisms and detritus. It is, in fact, formed by the coalescence of the small yellow patches to which reference has already been made; it does not extend beyond the tonsils, from which it can be easily scraped off without producing any hæmorrhage. It must again be repeated that this exudation may contain diphtheria bacilli, and the case go on into one of true diphtheria. Still the diagnosis of this disease from lacunar tonsillitis is, on the whole, much easier in the presence than in the absence of a membrane. In scarlet fever a membrane is rarely seen. When present it is of a dirty gray colour and seldom spreads beyond the tonsils; it has a great tendency to leave deep and chronic ulcers. Non-diphtherial membranous pharyngitis is a very rare disease, occurring mainly as a complication of influenza; it can only be distinguished from diphtheria supervening upon the latter by the associated symptoms and bacteriological findings. When the pharyngitis of measles becomes membranous it usually means that diphtheria has also set in; the same may be said of membranes appearing late in the course of scarlet fever or typhoid.

The implication of adjoining structures may also be a valuable help to diagnosis. The glands at the angle of the jaw are typically much less swollen in diphtheria than in scarlet fever or tonsillitis; the swelling is particularly marked in the last-named affection. But one occasionally sees cases of diphtheria—almost always of a very severe type—in which the glands are enormously enlarged. Implication of the nose is commoner in diphtheria than in either of the other conditions; a profuse nasal discharge is of frequent occurrence, and often contains the Klebs-Loeffler bacillus. It may also occur in scarlet fever, but the micro-organism is, in an uncomplicated case, absent. In the latter disease, on the other hand, epistaxis is much more likely to be present than in diphtheria. Neither discharge nor epistaxis is customary in simple tonsillitis. Scarlet fever is liable to cause inflammation of the middle ear;

neither of the other faucial inflammations under consideration spreads in this direction, with the rarest exceptions. Diphtheria, however, may at any time attack the larynx, which escapes implication in scarlet fever and simple tonsillitis. Finally, the tongue in scarlet fever, with its red, enlarged papillæ protruding through a creamy fur, is absolutely characteristic; this phenomenon is, however, inconstant though frequent.

"The strictly scarlatinal factor in the throat of scarlet fever," says Dr. Caiger (*Clinical Journal*, 31st Dec. 1902), "is a vivid red injection of the whole of the fauces, palate, and buccal mucous membrane. There may be much, or little, if any, swelling or ulceration of the tonsils; the amount is, as it were, purely a matter of accident in respect to the diagnosis of scarlet fever. The diffuse redness of the palate and buccal surface fades rapidly."

Associated symptoms and physical signs.—The *onset* of diphtheria is usually insidious, of scarlet fever and lacunar tonsillitis acute, the former usually with nausea or vomiting; definite influenzal symptoms, particularly in one who is subject to this affection, will point to simple tonsillitis.

The *temperature* is, as a rule, much higher in the other two diseases than in diphtheria; 104° or 105° is very seldom reached in the latter, quite commonly in the former, in which, moreover, it rises with great rapidity. It will be remembered that in some grave cases of septic pharyngitis the temperature may be low—even subnormal—throughout.

The *face* in scarlet fever is flushed and the *skin* markedly hot and dry. A *rash* is characteristic of scarlet fever, and when typical is not to be mistaken for anything else. Fallacies may, however, occur here. In the first place the rash of scarlet fever is very often atypical in character and especially in situation. In the course of some years' experience at the Evelina Hospital the writer has seen scarlatinal rashes commencing on almost every part of the body, including the forearms, legs, and buttocks. It is not infrequently a simple erythema, in which the typical punctæ are with difficulty or not at all to be detected. These facts will account for many of the instances in which the diagnosis is not made until desquamation has set in or other children have been infected. On the other hand, an erythematous eruption—quite apart from antitoxin rashes—may occur in diphtheria and lead to a branny desquamation quite unlike the flaky peeling which characterises scarlet fever. Finally, as already mentioned, the two diseases may complicate each other.

The examination of the *urine* is of great importance. Actual nephritis, with casts and especially with œdema, during the first few days is almost certainly diphtherial. In both the other conditions albuminuria frequently occurs early, but is merely febrile and in proportion to the temperature. The presence of albumin with a subfebrile temperature is thus a suspicious sign. Scarlatinal nephritis does not come on till about the third week.

The *knee-jerks* should be examined in all cases. If they are absent the disease is almost certainly diphtheria. In a daily investigation of over 150 cases of this affection, it was found that in quite 80 per cent they were absent at some period of the illness, if only for a day or so; they were absent throughout in about 10 per cent. The writer is thus led to place great reliance on their absence as a diagnostic sign, while admitting that their presence is not significant.

The *pulse* is apt to be slower, both absolutely and in relation to the temperature, in diphtheria than in scarlet fever or lacunar tonsillitis. A very slow pulse in association with sore throat is at the same time a help to diagnosis and an evil portent.

Dr. Caiger (*loc. cit.*) in doubtful cases of sore throat regards as one of the most distinctive indications of the scarlatinal nature of the attack, the appearance of *pain with tenderness* and more or less stiffness of *the joints* of the metacarpus, fingers, or wrists during the latter half of the first week. It may be so little marked as not to be complained of, but it is constantly present to some extent.

The *presence of membrane elsewhere*, as on the vulva or the surface of a wound, is of rare occurrence, but an almost certain sign of diphtheria.

Bacteriological examination.—Herein lies the final test, though it must, unfortunately, be confessed that bacteriology has not proved quite so serviceable as an absolute criterion as was originally anticipated. If the patient's throat contains virulent diphtheria bacilli he should obviously be treated for diphtheria, whatever his symptoms may be. But it is now established that the Klebs-Loeffler bacillus may be present for months together in the throats of healthy persons who have never had diphtheria. Perhaps these bacilli are not active, but to inoculate a guinea-pig—the sole test—takes time, and even then we are not certain whether virulence for the guinea-pig and for man mean the same thing. Moreover, increasing knowledge has added to the complexity of the differentiation of micro-organisms, and one has usually to be content with the statement, the day after the patient is first seen, that the

bacteriologist has succeeded in cultivating microbes morphologically indistinguishable from the diphtheria bacillus. This brings us back to the necessity for inoculation, which, as it is essential that antitoxin should be administered to a diphtheria patient without delay, is here inadmissible. It thus becomes necessary for the clinician to formulate a practical rule for his own guidance. Infallibility is obviously out of the question; what is needed is the nearest approach to accuracy which is workable. The writer's practice is as follows:—A swab is taken of every suspicious throat and stained at once by Gram's method. If bacilli resembling the diphtheria bacillus are present in any number—*e.g.* if one is seen in each field—the patient has an injection of antitoxin. Whether this be given or not, cultures from the throat are made upon blood serum and examined in twelve to eighteen hours. If a growth is seen it is stained by Gram's or Neisser's method, and if it consist—as it is most likely to do after so short an incubation—of bacilli like that of Klebs-Loeffler, the case is treated as one of diphtheria. The method is on the whole trustworthy. The patient may be wrongly suspected owing to the presence of non-virulent bacilli, or of the pseudo-diphtheria (Hoffmann's) bacillus, and he may have an unnecessary injection of antitoxin; but these drawbacks are far outweighed by the fact that "latent," atypical, or secondary (to simple tonsillitis) cases of diphtheria may be thus detected and treated without a delay which is sometimes fatal. It need hardly be said that a careful examination of the patient is always made according to the lines already laid down.

Such then are the general rules for the diagnosis of acute throat affections. Parenchymatous tonsillitis has hardly been referred to in them, as it is usually detected with the utmost ease. But it must be remembered that diagnosis is only a step, the chief one, it is true, on the road to treatment. Nor is it sufficient for this purpose to be content with the recognition of, say, lacunar tonsillitis. Many acute disorders, such as rheumatism and influenza, may express themselves in this fashion, or the throat condition may be the sole reaction of the organism to a noxa. Further investigation will be required before a definite conclusion and a sound basis for treatment can be reached. And it must above all be remembered that many acute throat cases may be of the utmost gravity. Children may and do die from cardiac failure of diphtherial origin after an apparently trivial "cold in the throat"; and a seemingly simple pharyngitis in an adult may suddenly develop into the most awful type of pharyngeal phlegmon. There is no region of the

body in dealing with which the practitioner has more need to be safe in diagnosis, guarded in forecast, and prompt in treatment.

SPECIFIC INFLAMMATIONS OF THE PHARYNX

Tuberculosis.—The pharynx may be involved in acute miliary tuberculosis, or be the seat of more chronic tuberculous changes. Primary tuberculosis of this region is distinctly rare. During the last ten years great stress has been laid upon the danger of infection through the tonsils. The alarm thus excited would seem to be greatly exaggerated. The tonsils are sometimes found to contain giant cells in cases of advanced phthisis, but the demonstration of active bacilli in their substance has but rarely been effected; in many instances they have no doubt become infected through the sputum. In a certain number of cases tonsillar tuberculosis is seen post-mortem, while the rest of the body has escaped and the patient has died of something else. Here in all probability the defensive activity of the tonsil has prevented a general infection. Lastly, there is a small proportion of cases in which it appears that the cervical lymphatic glands have become infected through the tonsils. It would hence appear that while recent research has been of value in directing attention to the tonsils as a possible channel of tuberculous invasion, there is no established evidence that such invasion is of frequent occurrence.

The less uncommon form of pharyngeal tuberculosis is characterised by the appearance of purple nodules along the edges of the posterior faucial pillars, more often on the right side than on the left. These nodules are tender, but at first cause very little pain; after a while they break down into indolent ulcers, with irregular edges. There may be enlargement and tenderness of the cervical glands. If the tonsils are implicated multiple ulcers are usually seen upon them, covered with a whitish slough, which may contain tubercle bacilli. The physical signs of phthisis will almost always be found in the lungs. Miliary tubercle of the pharynx is even more rare, and is scarcely ever primary.

Symptoms.—There is usually severe pain with hyperæmia, which on the appearance of the tubercles gives place to anæmia. The tubercles are at first pinkish, but soon become grayish yellow; they tend to fuse and break down, forming irregular superficial ulcers. The soft palate becomes infiltrated and immobile; the voice is nasal, and fluids regurgitate through the nose. There is

much pain both on coughing and swallowing, and the patient is at great discomfort owing to the accumulation of the sticky discharge from the ulcers; small wonder that he rapidly goes downhill. If the lungs were not involved from the first they will certainly be affected before death occurs.

Treatment.—The miliary form is inevitably fatal, and can only be palliated by the use of cocaine and morphia. An attempt may be made to eradicate the chronic nodules or ulcers by repeatedly cauterising with 50 per cent lactic acid, under cocaine; they can thus in some instances be destroyed and replaced by cicatrices. But, as already stated, pharyngeal tuberculosis is seldom aught but a phase in the downward course of phthisis.

Lupus.—In about 4 to 5 per cent of cases of external lupus there is involvement of the pharynx. Primary pharyngeal lupus is very rare, not occurring, as far as the statistics show, in more than one case out of six or seven hundred. The disease generally appears at or before puberty, and is commoner in girls than boys. The onset is usually insidious, the throat being but slightly sore, with perhaps a little dysphagia. Examination shows the affected region to be hyperæmic and studded with nodules, which are at first hard and red, but afterwards assume the characteristic "apple-jelly" appearance. The surface is much deformed and distorted; after a while a sluggish ulceration supervenes and proceeds at one spot, while cicatrisation is going on elsewhere. There is still little or no pain, and the absence of this, together with the occurrence of scarring, will usually mark the distinction from tuberculosis. The ulcers of lupus are, as a rule, somewhat prominent, with irregular, well-defined edges, and a dry base, looking not unlike dry velvet. Owing to its comparatively painless course the disease may have got a firm hold before it is detected, and may eventually erode the soft and hard palates, leading to both deformity and discomfort.

Treatment.—Isolated nodules can be scraped or cauterised, but the disease if extensive calls for the light treatment, the results of which are in the highest degree encouraging. The patient's general health will, of course, require support.

Syphilis.—Syphilis, both inherited and acquired, frequently attacks the pharynx; simultaneous manifestations of the disease in other parts are rarely absent.

Primary.—The hard chancre is seldom seen in the pharynx. It forms an indolent, sharply-cut sore, usually situated upon the tonsil, which is much indurated, and may be of stony hardness. The

neighbouring glands enlarge and harden, and secondary manifestations soon develop to clinch the diagnosis.

Secondary.—*Erythema* usually appears within two or three months of infection, and is synchronous with the cutaneous maculæ or papules. It presents a bluish red appearance, is symmetrically disposed upon the soft palate and faucial pillars—seldom involving the tonsils—and has quite a sharply-defined edge. It causes a definite but not extreme sensation of soreness.

Superficial ulceration is an extremely common and quite early symptom. The ulcers are usually symmetrical and kidney-shaped, and situated upon the tonsils, though the soft palate may sometimes be alone attacked. The subjective symptoms are seldom severe; the affection, as a rule, disappears rapidly, but may pass on into a more chronic and painful form.

Mucous patches appear somewhat later than the other secondaries, and may persist for years. They are mostly symmetrical, slightly elevated, and of a bluish white colour, and are surrounded by a narrow zone of hyperæmia. Their appearance has been compared to that produced by the application of lunar caustic to mucous membrane.

Tertiary.—The essential tertiary manifestation is the gumma. This not infrequently attacks the pharynx or tonsils, but as it produces little or no discomfort, is seldom seen until it has broken down and formed an ulcer. Before this has happened it appears as a smooth, rounded, often uneven, swelling, covered by hyperæmic mucosa, in which yellow spots, indicative of necrosis, soon appear. The ulcer is deep and cup-shaped, with steep edges and a sloughy, yellowish gray base. The soft palate is frequently attacked, and as the ulceration is virtually painless and extremely rapid, perforation may occur before the condition is recognised. In neglected cases the velum, and even the hard palate, may be completely destroyed. Finally, the healing of tertiary ulcers invariably leads to scarring; the cicatrices are stellate and radiating. The uvula may be bound down to the posterior wall, or bridges of scar tissue thrown out in all directions.

The **treatment** is that of syphilis in general. Mercury should always be given, as well as iodide, in the tertiary stage. Frequent recurrences may render it advisable for the patient to undergo a course at Aachen. Locally, nothing more than a weak gargle of corrosive sublimate is, as a rule, called for. Cicatricial stenoses may indicate surgical interference.

CHRONIC DISEASES OF THE PHARYNX

Chronic Pharyngitis.—It is stated that this may be of congenital origin, in association with general hypertrophy of the lymphoid tissue of the naso-pharynx. It is more often the result of repeated attacks of the acute variety, or it may follow other throat affections, such as those of measles, scarlet fever, and diphtheria. Certain general conditions predispose to it; among these may be particularly mentioned anæmia, rheumatism, gout, and gastric disorders, especially if associated with constipation. The principal excitants are excess in smoking, the abuse of spirits, exposure to irritating dust, as in the case of stone-masons, and habitual overstrain of the voice, particularly if the method of production be improper. Some degree of pharyngitis is a common symptom of chronic poisoning by arsenic, or by coal-gas.

Symptoms.—The patients, who are mostly by way of using their voices professionally, usually complain of some throat-ache, and of loss of timbre after speaking for some time. Singers and street-criers find that their higher notes become defective. As a rule there is a considerable accumulation of mucus, leading to hawking and even retching; the result is known to domestic pathology as a "stomach cough."

Morbid appearances.—These may vary considerably with the stage, and perhaps the cause of the affection. The simplest are those of a diffuse catarrhal inflammation, purplish in colour, intersected by dilated venules, and partly obscured by exudation of mucus. In the so-called granular form the pharynx is studded with little reddish nodules, representing hypertrophied lymph-follicles. The secretion is less than in the catarrhal form; the uvula is usually long and causes much coughing. An atrophy of the pharyngeal mucous membrane (pharyngitis sicca) may eventually develop, in which the pharynx is bare, glistening, and dry. This condition is not infrequently associated with atrophic rhinitis.

Treatment.—The primary anæmia, diathesis, or bad habit, must, of course, be first attended to; a change of scene and climate is often of great value. Locally may be employed a gargle or spray containing 2 parts each of borax and bicarbonate of soda, and 1 each of common salt and potassium chlorate in 100 of distilled water. For the cough, a lozenge containing $\frac{1}{4}$ grain codein, or $\frac{1}{8}$ grain heroin, may be sucked at night and repeated if necessary. The hypertrophied lymphoid follicles should be touched

with the galvano-cautery, which should also be applied to the dilated veins. The affection is likely to recur.

Pharyngomycosis Leptothricia.—Invasion of the pharynx by the leptothrix buccalis is not an event of extreme rarity. The growth may appear round the root of the tongue, and extend up the pillars of the fauces, or may be limited to circumscribed patches on the tonsillar crypts; the former variety was at one time thought to be of the nature of a seborrhœa.

Symptoms are very few. The patient is usually in poor health and may complain of some faucial dryness and huskiness.

White adherent growths are seen upon the affected parts; they look like the casts of a minute worm. There is no inflammation around, and threads of the leptothrix can be detected with the microscope.

The **treatment** is to improve the patient's general health, and to paint the growth with a 2 to 3 per cent solution of cresol, or a 1 in 1000 solution of perchloride of mercury.

Chronic Lacunar Tonsillitis. — Now and then an acute lacunar tonsillitis fails to clear up, but leaves the mucous membrane persistently inflamed; in such cases acute exacerbations are extremely frequent. The tonsil need not be enlarged, though a certain degree of hypertrophy is usually present. The lacunæ are apt to be deepened and choked up by cheesy plugs, containing fatty acids, débris of cells, and fungi. These plugs may decompose, rendering the breath noisome. The proper treatment is to squeeze them out, after nicking the edges of the lacunæ with a bistoury, if necessary, and then to apply astringent or antiseptic paints to the tonsils.

Tonsillar Hypertrophy.—This exceedingly common affection may be due to a variety of causes. It is at times congenital; it may be the residuum of an imperfectly resolved acute inflammation; or it may betoken a certain degree of chronic parenchymatous inflammation. The large tonsil is composed in the main of lymphoid tissue; when the normal process of atrophy has supervened in such an one it may be found still above the natural size, but stony hard, offering great resistance to the guillotine. Enlarged tonsils are most commonly found in children, though no age is exempt from liability to them. They are often complicated by the presence of adenoid vegetations. Fungus-like bodies (blastomycetes) have been described in them, but are of unknown significance.

Symptoms.—The parents' attention is most often called to the child's throat by the fact that it snores at night, or has difficulty

in swallowing. Growth is apt to be impeded, the child appearing stunted and anæmic. The voice is usually weak and nasal, and the hearing is often defective. This may be due to occlusion, with consequent catarrh, of the Eustachian tube. The deafness may affect one side or both. Dry, persistent, ineffectual cough is often present, and may be very worrying. There is great liability to repeated attacks of acute sore throat. After a while deformities may develop. The palatine arch tends to become pointed, and the line of the front teeth irregular; the child assumes the characteristic aspect of the mouth-breather. Later on it may become pigeon-breasted, or the chest may show a great deepening of Harrison's sulcus at the level of the upper edge of the liver. All these sequelæ are much intensified if, as is often the case, adenoid vegetations are also present.

Treatment.—It being recognised that symptoms such as those just described are due to tonsillar hypertrophy—and the large, pale tonsils are, as a rule, very easily seen—operative procedure is in almost every case indicated. It may be necessary, but is usually inadvisable, to remove the tonsils while inflamed; in mild cases touching with a pencil of silver nitrate will perhaps suffice. But in most cases the indication is as clear as the procedure is simple. The tonsil is pushed inwards by a finger on the neck, and cut off with a guillotine. The pain, such as it is, may be in great measure prevented by previously painting the surface with a 10 p.c. solution of cocaine, or deadened by sucking ice, and the hæmorrhage can be checked by the same means. Sometimes, especially in adults, the bleeding may be severe, even calling on one or two occasions for ligation of the common carotid; it need hardly be said that tonsillotomy should not be done in hæmophilic subjects. After the operation soft and non-irritating food should alone be taken for a day or so. When the tonsils are not readily brought into view, especially in older subjects, they may be burned with the galvano-cautery in preference to being cut; and in cases where they are very hard, it may be necessary to operate with the knife instead of the guillotine.

Tonsillar Calculi.—Calcareous concretions sometimes form in the tonsils. They are usually small, but may attain a length of $1\frac{3}{4}$ inch and a weight of nearly an ounce. These large stones are as a rule unilateral. They consist chiefly of the phosphates and carbonates of lime and magnesia. A few have been found to be composed of urate of soda. They have a peculiar evil smell, comparable to that of decaying cheese. Most of them are probably formed as the result of the growth of fungi, particularly leptothrix;

it has been alleged, though without proof, that some may result from calcareous deposition in retained pus. The so-called epithelial pearls occasionally met with in the tonsils appear to be the origin of some calculi. As a rule they produce no symptoms, but they may lead to acute or persistent attacks of tonsillitis or to suppuration. In the latter event the calculus may slough away and be inspired into the larynx, threatening asphyxia.

If a tonsillar calculus is suspected it should be sought with a probe, and if found cut down upon and removed.

RETRO-PHARYNGEAL ABSCESS

This is most commonly found in young children, in whom it usually depends upon an inflammation of the lymphoid tissue between the pharynx and the bodies of the second and third cervical vertebræ. This inflammation may be "idiopathic," or may follow acute specifics, such as scarlatina or measles. Less often the abscess is due to injury or to caries of the cervical spine. It is almost always unilateral, rarely central. It bulges into the pharynx from behind; the glands at the angle of the jaw are swollen on the corresponding side. If untreated rupture into the pharynx may occur, or the pus may spread into the cellular planes. The most dangerous complication is involvement of the larynx giving rise to acute inflammation or œdema.

The principal **symptoms** are cough and dyspnœa, with some dysphagia. The head is usually kept fixed, especially if the cervical vertebræ are involved. The bulging swelling may be visible when the mouth is opened; it is usually easily detected by the finger in the throat, except in children, who may offer considerable resistance.

The **treatment** is to open the abscess in the pharynx or, if it points externally, behind the sterno-mastoid. The head should be kept low during the operation, so as to prevent the escape of pus into the larynx. Any dead bone must be removed if possible. Hot fomentations to the neck often afford some relief. Tracheotomy or intubation instruments should be kept in readiness.

Diseases of the Uvula

The uvula is implicated in many attacks of acute and chronic pharyngitis. When acutely inflamed it is apt to become œdematous and may swell sufficiently to call for scarification.

Elongated uvula is not uncommon, though perhaps hardly so frequent as is usually supposed. It may either be simply relaxed or congested and hypertrophied. Cough and impairment of voice —especially in singing—are the principal symptoms, and are in most cases as much due to the associated pharyngitis as to the condition of the uvula. The treatment must aim as far as possible at the underlying affection, and will hence be on the general lines laid down in connection with chronic pharyngitis. Now and then it may be necessary to cut off the tip of the uvula, which should be drawn forward by forceps for the purpose. The raw surface will then look backwards and will escape irritation by food, etc.

Diseases of the Lingual Tonsil

Laryngologists have dignified a small nodule of lymphoid tissue at the base of the tongue with the appellation "lingual tonsil." Lacunar and parenchymatous inflammations of it are described. It also may undergo chronic hypertrophy, so that it becomes of an appreciable size. This may occur in association with chronic pharyngitis or without obvious pathological cause. It leads to hawking and cough, with a chronic sense of fulness in the throat. It may be one of the causes of "globus hystericus"; it is certainly less uncommon in women than in men. It should be painted with iodine or removed by a special curette.

NEW GROWTHS OF THE PHARYNX

Non-malignant new growths of the pharynx are rare, but very varied in nature; most of them spring from the tonsil. Papillomata, however, which are by far the least infrequent, are exceptional in this respect, being generally attached to the edge of the soft palate, the pillars of the fauces, or the uvula. They present a characteristic cauliflower-like appearance. Adenoma, angioma, and fibroma may also occur, and Onodi has collected four cases of lipoma and fibro-lipoma. Cysts of the tonsil are also occasionally seen; their pathology is uncertain.

All these growths give rise to very few symptoms; there may be slight dysphagia, some interference with the action of the soft palate and with phonation. Fibromata and angiomata sometimes bleed, and the former are now and then painful.

If they give rise to discomfort the growths should be removed; this is facilitated in many cases by the fact that they are pedunculated.

Epithelioma of the Pharynx

This is almost always primary; it is commoner in men than in women; and it is rarely seen before forty years of age. Its chief seats are the junctions of the pharynx with the mouth and œsophagus, and the tonsil. It usually spreads rapidly to surrounding parts, such as the larynx and the tongue; the glands are early affected. The growth first appears as a wart-like nodule surrounded by a zone of hyperæmia. It soon ulcerates and then spreads with great rapidity in all directions. A large, deep, irregular ulcer with a hard base is thus formed, covered with a foul, mucopurulent discharge, and often surrounded by nodules of new growth.

Symptoms.—These may at first be very slight: a little dysphagia, some trifling pain, and, if the parts adjacent to the larynx are involved, hoarseness. If ulceration be delayed the enlargement of glands—earlier and more marked when the upper part of the pharynx is attacked—may be the first symptom. Pain, when present, is sharp and most marked on swallowing; it becomes much more intense when the growth begins to break down. There is now a foul discharge from the mouth; salivation becomes troublesome, and the appetite fails. The cancerous cachexia soon becomes established; the patient wastes, and life is rarely prolonged for more than a few months. If a large vessel be eroded severe hæmorrhage will occur. The hardness and rapid growth of the disease and its failure to respond to antisyphilitic remedies are, from the diagnostic point of view, its most important characteristics.

Treatment.—The knife affords the sole hope of relief; except in the case of a growth springing from the uvula the operation is always difficult, but, according to Semon, yields a rather more favourable prospect than is usually believed. Glandular involvement is a fatal sign. The sufferings of the dying can be palliated by hypnotics, analgesics, and antiseptic washes.

Sarcoma of the Pharynx

Of the various forms of sarcoma found in the pharynx lympho-sarcoma is the commonest, while the round-celled and melanotic varieties are the most dangerous. The growth is situate most frequently upon the tonsil. Lympho-sarcoma manifests itself either as a definite tumour or as an infiltrating growth. The rate of development varies greatly, but ulceration always takes place sooner or later. Till then there is but little pain, the sole symptoms

being interference with phonation and deglutition. At this stage the growth has a pinkish, succulent aspect; when ulceration has supervened some cicatrisation may occur, so that ulcers, scars, and nodules of new growth may all be seen together. Sarcoma of the pharynx, and especially of the tonsil, has a great tendency to develop behind the jaw, and may lead to a large, hard external swelling at the upper part of the anterior triangle of the neck. There is not infrequently fever; sometimes the growths may temporarily diminish in size. Septic pneumonia may be the final complication. Severe hæmorrhage now and then occurs.

Treatment.—Removal of the slowly growing forms while they are still circumscribed affords some hope of relief, but the operation is not unattended with risk, both immediate and remote. In one case in which the tonsil was removed for sarcoma the common carotid had to be tied to check the bleeding.

NEUROSES OF THE PHARYNX

The pharynx is supplied by the fifth, ninth, tenth, and eleventh cranial nerves, and may suffer as the result of lesions anywhere in their course. It is sometimes affected in disease of the higher regions of the brain or in the later stages of myopathies. Post-diphtherial pharyngeal palsy may be both sensory and motor, and is no doubt due to an actual degeneration of both sets of nerves. On the other hand, a large number of the neuroses of this region belong to the category known as "functional." Many of these are associated with changes in the sexual organs.

Motor (a) *Paralysis.*—The most common cause of this is post-diphtherial nerve-degeneration (so-called "neuritis"). It results also from acute or chronic changes in the vago-accessory nuclei, which give rise to the symptom-complex known as labio-glosso-laryngeal paralysis; the disease may occur alone or be a terminal complication of progressive muscular atrophy, to which the chronic form is strictly allied, the acute resembling rather acute anterior poliomyelitis. Various other bulbar affections, such as thrombosis, embolism, or meningitis, may produce similar results. Unilateral pharyngeal paralysis is described by Hughlings Jackson in association with that of the tongue and larynx, the trapezius and sterno-mastoid being also sometimes involved. Pharyngeal paralysis may also be of hysterical origin (see Vol. III., pp. 245 and 272).

The **symptoms** may be very slight if the paralysis be unilateral. The uvula is drawn towards the healthy side, on which the velum

palati is lower than on the paralysed side. Bilateral paralysis causes the voice to become nasal and leads to difficulty in deglutition, especially of fluids, which are apt to be regurgitated through the nose. This greater obstruction to the passage of fluids than of solids is very characteristic. The velum palati hangs down and the "pharyngeal reflex" is lost.

Treatment is that of the underlying condition. Locally galvanism is useful in post-diphtherial, faradism in hysterical cases.

(b) *Spasm.*—This may be tonic or—very rarely—clonic. Of the former the well-known "globus hystericus" is probably an example. Tonic spasm of the pharynx is nearly always "functional," though it is marked in hydrophobia, and may also result from local irritation, as in acute tonsillitis. Clonic spasm is here virtually always a reflex neurosis, though it may occur without obvious disease of the naso-pharynx. The contractions of the velum palati may lead to a clicking sound which is distinctly audible; clonic adductor spasm of the vocal cords has been observed in association.

The **treatment** is general—by tonics, food, and fresh air; local galvanism may be of use in allaying the spasm.

Sensory.—(a) *Anæsthesia* is most commonly post-diphtherial; it may also be due to pressure upon the glosso-pharyngeal nerve, or to hysteria.

(b) *Hyperæsthesia and paræsthesiæ* are chiefly found in women. They may accompany anæmia or chlorosis; they are comparatively common at or about the climacteric, and they may recur with each successive menstrual period. Hyperæsthesia is usually of a neuralgic type; the paræsthesiæ are multiform, comprising sensations of tickling, dryness, soreness, choking, and so forth. They vary in intensity from time to time; less often do they change in character. In making the diagnosis the utmost care must be exerted to exclude any commencing organic disease; the early stages of cancer of the pharynx may be characterised by symptoms such as those just described. In these functional cases objective signs are absent or entirely incommensurate with the symptoms.

Treatment of post-diphtherial cases has already been referred to; other organic lesions are but little amenable to remedies. The "functional" cases are often very obstinate, particularly those occurring at the climacteric. The general health must be improved. A visit to a foreign watering-place may be of use. Weir Mitchell's rest treatment is often effectual, especially in young patients.

Bertram Abrahams.

DISEASES OF THE ŒSOPHAGUS

The œsophagus is merely the conduit by which food passes from mouth to stomach. It is to all intents and purposes as much outside the body as is the skin; it plays no active part in digestion, and the investigation of its diseases is hence conducted upon purely physical lines, with little or no assistance from chemistry.

METHODS OF EXAMINATION

External inspection is of very little service, but when aided by palpation may lead to the detection of a diverticulum or of the enlarged glands which accompany carcinoma. Endoscopy—best by means of Rosenheim's instrument—is not difficult, and may yield useful information in inflammatory and ulcerative conditions. A gum-elastic sound, or better and safer, a soft stomach tube, is a far more valuable aid to diagnosis. It is advisable to use one of a fairly large size, and to lubricate it with milk or glycerine, or preferably to allow the patient to moisten it in his mouth. He should then sit well back in the chair with the neck craned a little forward, the mouth open, and the chin tilted upwards and protruding; the sound is held penwise, about six inches from its tip, and passed along the roof of the mouth till it reaches the posterior wall of the pharynx. The patient is instructed to swallow when he feels it in this position, and with his co-operation there is seldom the slightest difficulty in the further passage of the instrument. The average distance of the cardiac opening from the teeth is 16 inches, and of the commencement of the gullet 6 inches. A superficial depression near the tip of the sound has been suggested with a view to scraping off a portion of any protruding growth. A more useful improvement is to have a few strands of lead wire woven in the apparatus, whereby its course can be rendered visible upon the fluorescent screen. The Röntgen rays are also of value in the localisation of foreign bodies impacted in the œsophagus.

Auscultation of the œsophagus is but little practised. The entrance of food into the stomach can be heard through a stethoscope, placed to the left of the spine near the tenth rib, in about six seconds from its leaving the mouth. Absence of the characteristic sound or great delay in its appearance would point to œsophageal obstruction. Sometimes the fluid may be heard to impinge upon an obstruction and then to trickle through (Kussmaul's sign).

MALFORMATIONS OF THE ŒSOPHAGUS

These may be congenital or acquired. It happens rarely that the œsophagus is at birth found to be divided into two segments, the upper ending blindly near the level of the tracheal bifurcation, the lower leading from the inferior part of the trachea to the stomach. The defect arises from a secondary obliteration of the œsophagus opposite the pulmonary diverticulum, and results in death from starvation within a few days.

Acquired malformations are of much greater frequency. They comprise general and local dilatations and obstruction. The latter will be discussed in connection with stricture of the œsophagus.

General dilatation of the œsophagus.—This is but rarely secondary to stricture; in such cases the symptoms are subsidiary to those of the primary affection. Primary dilatation occurs chiefly in adults. No satisfactory etiological explanation has been adduced, though an antecedent injury has been recorded in some cases. The dilatation is spindle-shaped, being greatest about the middle of the gullet; the muscular wall is hypertrophied and the whole organ usually elongated and somewhat twisted. A circumference of 6 inches has been seen.

The **symptoms**, which may persist for years, are dysphagia, pain in the chest, and occasional regurgitation of largish quantities of undigested food. The breath is foul and the tongue coated. The diagnosis from local dilatation (diverticulum) is of importance since the latter may be amenable to surgical treatment.[1] Rumpel's "two-tube test" is thus performed. Two œsophageal tubes are passed side by side. The one, which reaches into the stomach, has numerous holes in the course of its lower third; the other, which enters the pouch or dilatation, has a single terminal perforation. Water is poured into the former; in the case of a dilatation it cannot be withdrawn through the latter till the stomach is full; in that of a pouch it can be so recovered before any enters the stomach. Einhorn's "coffee experiment" is, if successful, a positive test for the presence of a pouch, which, however, cannot be excluded by its failure. A cup of black coffee is drunk by the patient, who makes every effort to force it into the stomach. An hour later he takes a glass of water. A tube is then passed; from a pouch clear water should be withdrawn, while on pushing the tube into the

[1] An excellent account of this will be found in a paper by Mr. James Swain, M.S., F.R.C.S. (*Brit. Med. Jour.*, 16th November 1901).

stomach a dark fluid would be obtained. In the case of a general dilatation the fluids would be indistinguishable. In this condition the swallowing sound above described is said to be absent.

Treatment is unsatisfactory. The food should be mainly fluid and given in small quantities at frequent intervals. Its administration through the stomach tube as a rule helps but little. Some relief may be afforded by lavage of the dilatation.

LOCAL DILATATIONS OF THE ŒSOPHAGUS — DIVERTICULA — 1. *Pressure or pulsion diverticula.*—These occur posteriorly, at the level of the cricoid cartilage. They really arise from the lower part of the pharynx, into which they open by a longitudinal slit, but do not cause symptoms till they press upon and obstruct the œsophagus. They consist of thickened mucous membrane covered with connective tissue; their walls contain no muscle fibres. They are usually found in males of middle or even advanced age. It has been suggested, on very insufficient grounds, that they are of congenital origin. Zenker and von Ziemssen hold, with much more probability, that they are due to trauma acting upon a part of the tube which is from anatomical causes weaker than any other.

Symptoms may be absent, the condition being detected only after death. When present the principal are increasing dysphagia, often associated with paroxysms of coughing, regurgitation of undigested food, and sometimes swelling of the neck. It is not infrequently possible to empty the pouch by pressure from without. The sound may be arrested in the pouch, a point which should be confirmed by the use of the fluorescent screen, or may at other times pass on into the stomach. Rumpel's and Einhorn's tests should be tried in doubtful cases. Patients in whom the cause of the dysphagia has not been diagnosed have died from starvation.

The **treatment** is to excise the pouch, an operation which was first performed by von Bergmann and has been strongly advocated by Mr. Butlin.

2. *Traction diverticula.*—These are usually from the anterior or side wall of the œsophagus about the level of the tracheal bifurcation. They result from the contraction of scar tissue, as in the case of adhesions round an inflamed bronchial gland. They are funnel-shaped, and from half an inch to an inch in depth. They cause no symptoms unless ulceration take place, which may be the result either of the original disease or of the pressure of retained food. In such an event a tracheo- or broncho-œsophageal fistula may follow and lead to septic pneumonia; in other cases empyema and mediastinal abscess have been the consequences. As the diagnosis

is made, if at all, after these complications have occurred, the question of treatment has not yet come under discussion.

INFLAMMATION OF THE ŒSOPHAGUS

Œsophagitis is a somewhat rare affection, many of the varieties being mere pathological curiosities, offering in respect to frequency a marked contrast to gastritis or pharyngitis. The following classification includes all the principal forms that have been described :—

A. Acute inflammation :
   I. Primary.—1. Catarrhal ; 2. Follicular ; 3. Necrotic ; 4. Phlegmonous.
   II. Secondary.—1. Mycotic ; 2. Diphtherial.
B. Chronic inflammation :
   I. Simple.
   II. Specific.—1. Tuberculous ; 2. Syphilitic ; 3. Actinomycotic.

Acute Œsophagitis

I. Primary—1. *Catarrhal.*—Inflammation spreads but rarely from the stomach, pharynx, or trachea to the œsophagus. The commonest causes of acute catarrhal œsophagitis are direct irritation and infectious diseases. Irritation may be due to contact with hot or corrosive liquids, neat spirits, or to the rough use of the sound ; the principal acute specifics in which the œsophagus may be inflamed are measles, scarlet fever, and typhoid. Smallpox may even give rise to a pustular eruption in the gullet, an appearance which has also been stated to follow the excessive use of tartar emetic. A true primary or "idiopathic" œsophagitis appears to occur in suckling infants.

The most marked **symptom** is ill-localised pain, greatest on swallowing or bending the neck. Thirst is common, as also is the eructation of a tenacious stringy secretion sometimes tinged with blood. The mucous membrane of the œsophagus is reddened and swollen ; the surface may be covered with a whitish fur, or eroded or even ulcerated. The irritation may lead in a predisposed subject to spasmodic stricture.

Sequelæ.—The affection may assume an exfoliative character by spreading to the submucosa. This is usually a grave condition ;

the mucous membrane may be brought up in long tubes. In other cases the inflammation becomes chronic. Traumatic cases are apt to go on to fibrous stricture.

The **treatment** is to rest the œsophagus by rectal feeding in severe cases, liquid diet in slighter ones; pain is to be relieved by morphia internally and belladonna fomentations externally. The administration of appropriate antidotes is an obvious indication after the swallowing of corrosive fluids.

2. *Follicular.*—This may complicate acute or, more often, chronic œsophagitis, or arise *de novo*. The inflammation affects mainly the glands, which become swollen and may even form little submucous retention cysts. In severe cases the formation of localised ulcers or abscesses is recorded. No satisfactory treatment is known.

3. *Necrotic.*—This serious form of œsophagitis is most likely to occur in the course of an acute toxæmia, as in smallpox, scarlet fever, typhoid, and cholera. It is found also in uræmia, and may complicate a necrosing pharyngitis. The symptoms are severe; if, as sometimes happens, a fibrinous pseudo-membrane is formed, shreds of it are brought up; if ulceration occurs, hæmorrhage is often profuse. In the latter class of cases stricture may follow.

4. *Phlegmonous.*—This may follow the simple form or arise from similar causes. It may be circumscribed or diffuse; the latter is the rarer and severer condition. Pus-cocci are present in both cases, and the affection may be regarded as a catarrhal œsophagitis complicated by them. Dysphagia appears to be an inconstant symptom, but pain, fever, and the vomiting of pus are usually present. In the "dissecting" form of this affection casts of the œsophagus may be brought up. The prognosis is grave and the diffuse form is almost always fatal. Cases caused by the impaction of a foreign body have recovered after its removal.

II. Secondary—1. *Mycotic.*—Quite a number of cases are known in which thrush has extended from the mouth down into the œsophagus. In most of these the diagnosis has not been made, but the extension has sometimes been suspected by reason of increasing dysphagia. The writer is not aware of any recorded case in which recovery took place.

2. *Diphtherial.*—In diphtheria the œsophagus usually escapes, even in those rare cases in which the stomach is attacked. Still diphtherial œsophagitis is described as having been diagnosed and even successfully treated.

Chronic Œsophagitis

I. Simple.—This is by no means uncommon, and is not infrequently found unexpectedly at autopsies. Heavy drinkers and smokers are particularly liable to it; it may also result from the chronic venous congestion of old-standing heart or lung disease. It naturally complicates stricture or dilatation of the tube.

The mucous membrane is pale and thickened, sometimes passing on into a condition comparable with that known in the mouth as leukoplakia. This has been seen in cases of malignant stricture of the œsophagus, but whether as a cause or a complication is not as yet certain. In some cases the walls are weaker as the result of the morbid change and favour the occurrence of dilatation.

The only marked symptom of chronic œsophagitis is a moderate degree of dysphagia; the pale mucosa with dilated venules and clammy secretion is not difficult to recognise with the œsophagoscope.

If local medication be required, silver nitrate or tannic acid can be introduced by means of an appropriate syringe after the sensitiveness of the organ has been reduced by cocaine or eucaine.

II. Specific—1. *Tuberculous.*—Kraus recognises six groups of causes of this rare condition. These are direct infection from swallowing tuberculous material, extension from neighbouring organs, bursting of caseous bronchial or mediastinal lymphatic glands (the least infrequent), of prevertebral abscesses or of pulmonary cavities, and finally miliary tuberculosis. The complication may be without symptoms, though pains in the back and sternal region with dysphagia may be present; its diagnosis is uncertain and its treatment merely palliative.

2. *Syphilitic.*—This is less rare than the last, and is the cause of an appreciable number of cases of œsophageal stricture, though perhaps not so many as were formerly attributed to it. Secondary inflammations may spread down from the pharynx, but are of no importance. Tertiary ulcers, which are of course very likely to cicatrise, occur only in subjects who are obviously gravely infected, and the resultant dysphagia is easily referred to its right cause. There is no special treatment; cicatricial strictures call for dilatation by the sound, or if that fail gastrostomy.

3. *Actinomycotic.*—Garde has recorded four cases of primary actinomycosis of the œsophagus.

A chronic œsophagitis is one of the characteristic phenomena of sprue.

SIMPLE ULCER OF THE ŒSOPHAGUS

The œsophagus may be ulcerated as the result of injury, of syphilis, of tubercle, of malignant disease, of the pressure of foreign bodies, and from other definite causes, but when all ascertainable etiological factors have been excluded, a certain proportion of cases remains which must perforce be grouped under the heading of simple ulceration. In the greater number of these the ulcers are situate in the lower third of the gullet, and resemble in anatomical characters the ordinary gastric ulcer. It is hence reasonable to suppose that the two are also pathologically related, and there is indeed evidence that some cases of œsophageal ulcer are associated with conditions, such as excessive dilatation of the stomach, which permit regurgitation of the gastric juice so that it may come in contact with the walls of the œsophagus. Such cases appear to be more common in men than women, and to occur chiefly in middle age; alcoholism, chronic cardiac and renal disease, and trauma are alleged to be predisponents. The ulcers are usually single; if they are multiple there is a tendency to confluence.

Ulcers of the upper part of the œsophagus are rare and of very obscure origin; they are probably induced by trauma acting upon a mucous membrane already weakened by chronic venous congestion. The vascular element evidently predominates where the œsophagus is, in common with the rest of the alimentary canal, the seat of multiple ulcers.

Simple ulcer of the œsophagus is rarely diagnosed during life; pain, spasm, dysphagia, vomiting, and especially hæmatemesis are the most significant **symptoms**. The pain is stated to be very characteristic. It is violent and burning, is increased by pressure, and radiates from the sternal or epigastric region into the shoulders, breasts, or hypochondrium. If blood be regurgitated it is usually fluid, and red or brownish in colour, unless it have first passed into the stomach.

The ordinary course of the affection is gradually downhill, the patient steadily weakening and wasting. The ulcer may cause rupture of the œsophagus or perforation leading to fistulæ into other organs. Sometimes healing occurs, and there is then a great liability to cicatricial stricture.

The ideal **treatment** is to allow the ulcer complete rest until it is healed. If the patient's condition permit, this is best effected by

means of gastrostomy. Failing this, rectal feeding must be resorted to. If cicatrisation produce narrowing, the lumen of the tube must be kept patent by means of bougies.

MALIGNANT DISEASE OF THE ŒSOPHAGUS

This is the commonest and most important of the graver diseases of the œsophagus. It attacks men about three times as frequently as women; more than 92 per cent of the patients are over forty years of age. Its actual causes are uncertain, but it is well known to be relatively common in alcoholic subjects, perhaps as a sequel to the chronic œsophagitis to which they are liable. It has been seen to develop in connection with œsophageal pouches, and from simple strictures.

Malignant disease of the œsophagus is almost always primary; epithelioma is the most common form, but glandular carcinoma and colloid are sometimes seen. Cancer of the cardiac end of the stomach, which produces all the symptoms of œsophageal disease, is of course derived from the gland-cells. Scirrhous development is frequent in all forms. About a dozen cases of sarcoma are on record. The progress of the growth is usually attended with ulceration and sometimes with scarring. Sarcomatous growths may manifest polypoid development. Malignant disease sometimes involves almost the whole length of the œsophagus. It is more often localised in certain spots, especially at the level of the bifurcation of the trachea, and at the two ends of the tube. In about four-fifths of the cases the growth commences in the middle or lower third; it is, however, curious that in women malignant disease seems to show a greater predilection for the upper third. It usually tends to grow, at first at any rate, in an annular direction, but may spread later both upwards and downwards. The growth remains at first limited to the œsophageal wall, and tends rather to obstruct the tube than to spread to surrounding parts. After a while, however, adhesions form to neighbouring organs, and the process of ulceration leads to the establishment of fistulous communications between them and the œsophagus. The trachea, either or both bronchi, the lungs—especially the right—or more rarely the pericardium, may be thus involved. An œsophageal carcinoma has been seen to spread into the left auricle; ulceration has also occurred into the descending aorta and the right common carotid artery, but these accidents are much rarer than the close

relation of the structures might suggest. The implication of the great vessels speedily results in fatal hæmatemesis. Extension into the pharynx or stomach is rare, into the connective tissue of the neck and posterior mediastinum much more common. In the latter case the growth may compress the recurrent laryngeal, vagus or sympathetic nerves, especially upon the left side. It has been found to implicate the vertebræ and even to extend into the spinal canal with resultant paraplegia. Metastases occur in at least 60 per cent of cases. The neighbouring lymphatic glands are most often affected, then the liver and lungs, sometimes the kidneys, bones, and spleen. It is not uncommon to find secondary growths studding the pleura and peritoneum. The extension of the disease into the air-passages is a common cause of death—from septic pneumonia, empyema, pulmonary gangrene, etc. As with other forms of œsophageal stricture, the tube is dilated to a variable extent above the lesion. Ewald records a case where the distension occurred below the stricture, and was due to weakening of the walls from chronic inflammation.

Symptoms.—The early symptoms are those of œsophageal obstruction, the later depend upon perforation into or implication of surrounding structures. Difficulty in deglutition almost always comes first, though where there is much ulceration there is sometimes but little dysphagia. The difficulty primarily affects solids, which after a while can only be swallowed if moistened, and eventually cannot be got down in any circumstances. The attempt at deglutition may sometimes be attended with pain; it more often induces a fit of coughing. The dysphagia is rarely of quite sudden onset. As it progresses the patient rapidly emaciates, as the result of starvation combined with the cancerous cachexia. Regurgitation of food is an early symptom. The time at which it follows a meal varies with the amount of dilatation which has supervened above the growth. It is at first usually painless, but may later on be accompanied by sensations resembling tormina. The material brought up is alkaline in reaction, unless, as I have not infrequently observed in early cases, the muscular effort of regurgitation induces an attack of true vomiting as well. Later on the regurgitated food is mixed with sloughy débris, which is usually fœtid and often blood-stained. Hunger and thirst are often early symptoms of the disease; later on all desire for food is usually lost.

On passing a sound the site and lumen of the stricture may be ascertained. In the commonest form of stricture the sound is arrested at the lower edge of the ulceration. A soft rubber tube

of considerably greater diameter than the sound can often be passed on into the stomach. The mucus with which the instrument is covered when withdrawn should be carefully examined; the characteristic cells of malignant disease are but rarely found, but when seen are diagnostic.

Those who are expert in its practice attach in this connection great importance to œsophagoscopy. According to them a malignant growth may be seen protruding into the lumen as a knob or prominence which tends to assume an annular form, and is immovable with respiration. Later on inflammatory and ulcerative changes can be seen in the mucous membrane. Auscultation merely confirms the fact of œsophageal obstruction, but does not help to elucidate its cause.

Among the later symptoms may be hiccough and post-sternal pain. There may also be evidences of extension, as for instance the presence of enlarged glands in the neck, hoarseness from implication of the recurrent laryngeal nerve, myosis, narrowing of the palpebral fissure, and retraction of the lids from involvement of the sympathetic; and eventually septic pneumonia. The secretion of sweat may be inhibited on one side; subcutaneous emphysema has been described as the result of ulceration into the lung.

The **diagnosis** will be considered under the general question of that of œsophageal obstruction.

The **course** is usually very rapid; cases in which scirrhous change is marked survive longer than others. The average duration when diagnosed is six to eight months; the longest which I have found recorded was in a case which lived certainly three and a half years.

Treatment.—It is obvious that no cure is to be hoped for but by excision of the growth. This is hardly ever practicable, but has been successfully performed on a very few occasions, though all the patients died of recurrence. Dilatation of a malignant stricture is an indefensible procedure; it increases the ulceration and may lead to secondary implantations. Feeding through a permanent cannula (such as a Symonds' tube) is hardly less unscientific, for the lumen of the gullet continues to grow narrower, the patient's discomfort steadily increases, and when a rational operation is at length undertaken it is almost always too late to be of service. The proper treatment of malignant disease of the œsophagus is by a palliative gastrostomy, undertaken at an early stage, while the patient is able to tolerate and benefit by it. I have seen several patients survive this operation for six or seven months with surprisingly little inconvenience; a duration of as long as two years has been recorded.

The patient's comfort is greatly increased if he be allowed to masticate his food before it is introduced into the gastrostomy tube; the researches of Pawlow show that the mastication of sapid substances is of value also as reflexly increasing the flow of gastric juice. If the patient refuses or is too weak for gastrostomy he will require at the last to be fed per rectum. When perforation into the air passages has occurred, oral feeding must of course be abandoned.

Non-malignant new growths of the œsophagus are mere curiosities. Myomata, fibromata, papillomata, and lipomata have been described, and are said in some instances to give rise to œsophageal polypi. Dermoids are also of great rarity.

Fibrous Stricture of the Œsophagus

As has already been indicated, the cicatrisation of œsophageal ulcers may lead to stricture of the gullet. It is convenient, if not very scientific, to consider all obstructions from this cause together, instead of under the headings of their various etiological factors.

Etiology.—The most frequent cause is trauma; a certain number of cases are the result of syphilitic ulceration; the remainder, which would once have been termed "idiopathic," are of obscure origin, though it cannot be doubted that some of them arise from the healing of the simple ulcers of the œsophagus, which have been described above. Trauma usually denotes the swallowing of corrosive fluids less often of foreign bodies, such as swords in the case of jugglers, or hair-pins in that of hysterical women. The fluids are mostly of a moderate degree of corrosiveness, insufficiently powerful to kill by their action on the stomach, but yet capable of producing severe œsophageal ulceration.

Symptoms.—It must be remembered that, as in the case of the urethra, and probably of the bile duct, a moderate degree of stenosis may be converted into complete stricture by the supervention of spasm. Moreover, a large food-bolus may stick in the narrowed channel with a similar result. The first evidence of the stricture may hence be a sudden inability to swallow, an inability which is too often called "functional," without investigation of its organic cause. More typically the dysphagia comes on gradually and without other symptoms, those of the precedent ulceration having perhaps entirely disappeared. In syphilitic cases other obvious evidence of the disease is almost always present; where trauma is the cause it is rarely difficult to obtain a history,

unless, as with would-be suicides, there is deliberate concealment. In the absence of ulceration there is little or no pain, nor is the vomit or bougie blood-stained. The sound is always arrested, and at the same spot; a coudé tube may pass where a straight one fails. There is some wasting, but not more than can be accounted for by the diminution in the amount of food utilised. Pressure symptoms are absent.

Treatment.—A cicatricial stricture of the œsophagus can be cured by dilatation with bougies of increasing diameter. The utmost patience on the part of operator and subject is required. In the case of obstruction near the cardia the stomach may be opened, and an attempt made digitally to dilate the stricture after the manner of Loreta's operation on the pylorus. As a palliative measure, or during the course of dilatation, the patient may be fed by gastrostomy or through a Symonds' tube.

The differential diagnosis of œsophageal obstruction.—Many conditions may be evidenced by dysphagia; their distinction is not as a rule difficult, provided a careful examination be made. It is first necessary to ascertain whether the obstruction is due to œsophageal or extra-œsophageal causes, though it may be observed that the latter rarely give rise to serious dysphagia, which usually implies paralysis of the peristaltic power of a considerable stretch of the tube. The careful examination of the chest should suffice to detect the presence of a compressing aneurysm or mediastinal tumour; in this respect some help may be afforded by the Röntgen rays. Sometimes the source of pressure baffles all research; Sir F. Treves tells me he has seen œsophageal obstruction due to retro-peritoneal sarcoma.

Every effort having been made to exclude extra-œsophageal causes, it remains to make the diagnosis between the various forms of œsophageal stricture. Those which have to be especially considered are spasm, cicatricial stricture, and malignant disease, and it must be particularly remembered that diverticula of the œsophagus usually first signalise themselves by obstructive symptoms. Of intrinsic causes of œsophageal obstruction malignant disease is the most common, its frequency being variously estimated at from 70 to 90 per cent of the whole; it is nevertheless to be diagnosed, in the early stages at any rate, mainly by exclusion. As to spasm, it is usually of sudden onset; but this, as has already been noted, is not pathognomonic, and, moreover, it has been pointed out above that spasm may complicate other œsophageal conditions. Of great importance is the fact that spastic obstruction is in general inter-

mittent, and that the passage of the sound is sometimes opposed, sometimes permitted. In these respects similar symptoms may be caused by a diverticulum; it must, however, be said that if the obstruction is always overcome by the sound the condition is one of spasm. The same is true of an obstruction which disappears when the patient is placed under an anæsthetic. Repeated trials with a sound will always hit upon a diverticulum sooner or later. In simple spastic obstruction there is no vomiting, and pressure symptoms are of course also absent. Œsophagoscopy reveals no morbid changes.

The special means by which the diagnosis of œsophageal diverticula may be established have been already referred to. When a history of trauma is present the diagnosis of cicatricial stricture is easy, particularly where wasting is inconspicuous; it is of course possible for such a stricture to become malignant, but this event must be of the extremest rarity. In doubtful cases German authors lay great stress on the evidence afforded by œsophagoscopy. The typical appearance in cicatricial stricture is said to be that of white scars—infiltration, or bulging of the wall inwards being absent. It must, however, be remembered that ulceration, as from malignant disease, may be concealed beneath what appears to be a simple constriction above.

In the case of malignant disease the family history of the patient is of great importance; the writer thinks that this can be obtained in quite half the cases of cancer of the œsophagus. The cachexia and wasting are here as a rule very marked, though this is not an absolutely trustworthy guide. Of more significance is the fact that the difficulty in swallowing is as a rule noticed at a period when no obstruction to the passage of the sound is present; *per contra* latent cancer of the œsophagus is very rare, though Dr. Emanuel has recorded an interesting series of six cases (*Lancet*, 18th October 1902). An otherwise inexplicable paralysis of the recurrent laryngeal may point to this affection; metastases in glands or liver are even more conclusive. With the aid of the œsophagoscope a piece of the growth may be snipped off for microscopical examination. Although obstruction is usual, in a certain number of cases of cancer of the œsophagus this symptom is wanting throughout, and the real cause of the patient's illness may be overlooked. Dyspepsia, nausea, and repugnance for food, and laryngeal paralysis, as well as enlarged cervical glands, may all be early symptoms and suggest the true nature of the case. Sometimes the diagnosis has not been made till the presence of cough after swallowing revealed

the existence of a communication between the œsophagus and air-way.

Simple (non-cicatricial) stenosis of the œsophagus is a curiosity described by Dr. Kendal Franks (*Brit. Med. Journ.*, 3rd November 1894) and others.

LESIONS OF ŒSOPHAGEAL CONTINUITY

Perforation.—The œsophagus may be perforated from within by instruments introduced for surgical or other purposes, or as the result of ulceration, particularly in connection with malignant disease and diverticula; or from without by the invasion of inflammatory processes in neighbouring parts. Except where there is actual trauma the perforation is but an incident—though one of the gravest significance—in the progress of an affection which has already been discussed. The symptoms of traumatic perforation resemble those of rupture. It is said that small perforations may heal; larger ones are ordinarily fatal from sepsis.

Rupture.—This is of rare occurrence; it can happen to a perfectly normal gullet, though in some instances the walls may have been weakened by the digestive action of regurgitated gastric juice or previous inflammation. Most cases seem to have occurred in drunkards. The cause is powerful contraction of the muscular wall, usually due to a violent effort to expel an impacted mass of food or other foreign body. Contraction of the diaphragm without vomiting was sufficient in one case. In every recorded instance but one the tear has been longitudinal; it is always situate in the lower third of the œsophagus.

The **symptoms** come on with great suddenness; there is often violent pain in the pit of the stomach, with a tearing sensation in that region, followed by profound collapse. Subcutaneous emphysema soon appears, especially in the supraclavicular regions; dyspnœa supervenes, the pulse-rate increases, and the patient usually dies within one to three days. Should he survive longer—the most protracted case recorded lived eight days—gangrenous mediastinitis and empyema follow. It is curious that immediately after the rupture there may be but little difficulty in swallowing fluids; some frothy blood-stained mucus is often brought up.

The **diagnosis** is difficult: the emphysema is a valuable sign, but will not distinguish rupture of a healthy œsophagus from perforation of a diseased one.

Treatment has hitherto been ineffectual. To give analgesics, to keep up the heart strength, and to feed per rectum, are the sole indications.

ŒSOPHAGEAL HÆMORRHAGE

Nothing need be said here regarding hæmorrhage from the rupture of an aneurysm into the œsophagus, or occurring in the course of malignant or inflammatory diseases of the tube, such as have already been described. Bleeding may, however, take place to an alarming and even a fatal extent from varicose veins in the lower part of the œsophagus. These, which are in relation to the portal system, may swell as the result of hepatic disease, particularly cirrhosis, but have also been found enlarged without assignable cause. The blood brought up is usually in large quantity, of a bright-red colour, and alkaline in reaction unless previously swallowed into the stomach. The patients are said often to be able to predict a hæmorrhage by the onset of premonitory symptoms, such as pain in the stomach, constipation, loss of appetite, and swelling of the spleen. Unless the dilated veins can be seen by œsophagoscopy the diagnosis is only to be suspected by exclusion.

NEUROSES OF THE ŒSOPHAGUS

The function of the œsophagus is to subserve the normal performance of what is known as the third stage in the act of deglutition. This consists in the involuntary transmission of a food-bolus by the peristaltic activity of the œsophageal musculature.

Virtually all the diseases of the œsophagus here discussed have manifested themselves by dysphagia, or, in other words, impaired or difficult deglutition. This may also result from affections, especially painful affections, of the mouth, tongue, or fauces, and from extra-œsophageal causes which have been enumerated in connection with obstruction of the gullet. But dysphagia may arise from purely nervous maladies, such as injury or disease in the province of any of the nerves concerned. And when all these causes have been excluded there still remains a class of cases which has to be considered under the heading "neurotic."

ŒSOPHAGEAL SPASM

"Idiopathic" œsophageal spasm—*i.e.* that in which a local exciting cause, such as ulceration, is not apparent—occurs chiefly in women. Out of forty-three cases of which I have notes only eleven were males. It may occur at any age from fifteen to fifty; in my series the average age of the women was twenty-five, of the men thirty-two. It has been met with in boys even as young as seven years. A psychical element is virtually constant in the etiology; the complaint may date back to some real or imaginary œsophageal injury. Thus a patient was seized, as the result of gulping a glass of champagne, with violent pain in the chest so that she fainted. From this time on she began to complain of increasing dysphagia; hers became eventually a typical case of spasmodic stricture of the œsophagus.

The principal **symptom** is dysphagia, which may come on gradually, but often appears in a severe form quite suddenly. Swallowing frequently evokes a sense of oppression in the chest, or even violent shooting pain. Solid food is sometimes swallowed better than liquid; the first mouthfuls are often rejected at once, while the later ones are more fortunate. Both these anomalies point strongly to spasmodic stricture. Occasionally the attempt at swallowing may lead to faintness or convulsions; I have seen it attended with a typical hysterical fit. Globus hystericus is not infrequently present.

An œsophageal bougie can usually be passed with ease if the patient's confidence has been gained; its use is often of great therapeutic value. Under chloroform there is no evidence of obstruction; unless unjustifiable force has been used the sound when withdrawn should not be blood-stained. Auscultation finds the entrance of food into the stomach to be inaudible or greatly delayed. The obstruction may be continuous, either remaining stationary or increasing in degree, or intermittent; I have twice seen it recur at each menstrual period. If the passage of an œsophageal sound is found to be arrested, the length that can be swallowed may often vary from time to time. In spite of the symptoms the patient seldom wastes, at least to any notable degree, although considerable emaciation has been noticed on first coming under observation.

The **treatment** is primarily that of the underlying neurosis. Tonics, sedatives, change of scene and climate, may all be of use.

Severe cases need the Weir-Mitchell procedure. The occasional—not too frequent — passage of a bougie may help. Electricity, preferably faradic, is of use chiefly from its "moral" effects.

ŒSOPHAGEAL PARALYSIS is as an isolated affection almost unknown, but may occur in conjunction with laryngeal and pharyngeal paralysis in hysteria, in severe brain injuries and diseases (*e.g.* shortly before death from hemiplegia), in bulbar palsy, tabes, disseminated sclerosis, and peripheral nerve intoxication by diphtheria, lead, alcohol, and syphilis.

The principal **symptom** is dysphagia; fluids pass into the stomach with a sort of rumbling noise (dysphagia sonora), while solid particles stick in the œsophagus and produce similar discomfort to that felt in actual stricture. They can, however, be usually washed down by a subsequent draught of fluid. There is but rarely regurgitation. The sound passes readily, and when in the œsophagus is susceptible of wide lateral movement.

The **diagnosis** must usually be inferred from the associated symptoms. In the absence of other paralyses this condition can hardly be distinguished from fusiform dilatation of the gullet.

Treatment must aim mainly at the primary affection; when this is an intoxication the prognosis is not unfavourable. Local measures, such as electrification, seem to be of little use, but the condition may be palliated by feeding through a hollow sound.

SENSORY NEUROSES. — Hyperæsthesia is not infrequently observed, but usually tends to lead up to and merge with œsophageal spasm. When it occurs alone a careful examination must be made to exclude an organic cause.

A well-known paræsthesia of the œsophagus is globus hystericus, a symptom of neurasthenia as well as of hysteria.

Anæsthesia of the œsophagus has been doubtfully described as a complication of diphtheria.

The **treatment** of sensory neuroses of the œsophagus is to, so to speak, wash it out by copious draughts of weak tea or soda water. Occasional wineglassfuls of a 1 in 3000 solution of silver nitrate, taken fasting, are also recommended.

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DISEASES OF THE STOMACH AND INTESTINES

In entering upon a consideration of the diseases of these organs it is necessary to remember that there are two groups of morbid conditions met with, which are more or less independent, at least in origin, though constantly associated, and to be investigated from different standpoints and by different methods. On the one hand are structural diseases of the organs, such as ulcer, inflammation, new growths, etc., whether primary in occurrence or determined by malnutrition of the body generally or by special disease of other parts, and characterised by certain signs and symptoms, local and remote, from which the nature of the lesion may be inferred. In connection with such diseases the digestive process will be likely to suffer more or less as the motor and secretory functions of the organs are interfered with by the morbid changes therein. On the other hand are perversions of digestion arising for the most part independently of previous structural change in the organs, and attributable in the first place to the qualities of the ingesta or the mode in which they are presented to the stomach from the mouth, to imperfections in the composition or quality of the digestive secretions, to inefficient or excessive motility, to nervous disturbances, or to other interferences with digestion that may be, primarily at least, due to causes apart from the organs immediately concerned. At the same time it must be admitted that the maintenance or constant repetition of such conditions may be the determining factors in the production of structural disease of the stomach or bowels. In approaching the subject of gastro-intestinal disease, therefore, the difference between indigestion or dyspepsia, and definite structural lesions of the tract, must to some extent be kept in mind in the proper analysis of the symptoms presented and in the pursuance of treatment.

A further general observation appears desirable. Whilst it is the diseases of the stomach and intestines and perversions of their functions that will be here described, the clinical necessity for their consideration in relation to other organs and other functions cannot be too strongly insisted upon, and especially for the formation of a prognosis and of a rational therapeutic plan. A too restricted view of gastro-intestinal disease, as indeed of any other organ, will be likely to furnish a very imperfect conception of the case as a whole.

General Etiology of Gastro-Intestinal Diseases

Affections of the stomach are amongst the commonest of maladies. So closely associated are its functions with those of other organs, that few diseases, whether acute or chronic, are not sooner or later complicated by gastric disturbance. Certain circumstances, however, would seem to confer a special liability, and so far may be regarded as predisposing causes, as others (exciting causes) are more surely responsible for the actual determination of gastro-intestinal disease.

Heredity.—How far any disease of the organs under consideration can be said to be the result of hereditary influence is exceedingly doubtful. Not infrequently, however, definite structural disease may so develop in several members of a family in the same or succeeding generations as to suggest an explanation of this kind. Dr. Dreschfeld (Allbutt's *System of Medicine*, vol. iii. p. 520) has recorded eight examples of gastric ulcer thus occurring. Still more numerous are the cases of cancer of the stomach, with a family history of malignant disease—14 per cent in 1744 cases as determined by Welch—and commonest of all is the existence of a "weak digestion" in parent and child. Dyspepsia of nervous causation seems specially liable to be transmitted. Notwithstanding the figures that can be adduced in support of this view, it is doubtful whether, when not merely coincidence, they are anything more than an expression of the hereditary transmission of a tendency due to a deteriorated resisting power on the part of the tissues concerned, and requiring for the development of the malady a special excitant, such as members of the same family are likely to be exposed to, whether this be habit of life, diet, or the like. Probably it is in this way that is to be explained the observation of Treves, that "perityphlitis runs often in families," and that it is less an inherited disease than a family one.

Age, as influencing the incidence of gastro-intestinal disease, is undoubtedly potent. Acute catarrhal affections are of great frequency at all ages, and children and infants are especially prone to suffer, the conditions being determined by comparatively slight causes, but it is to be noted that in such cases the intestine shares in the catarrh more frequently than in later life. The graver condition of phlegmonous gastritis is seldom met with except in adults, and cirrhosis ventriculi only in middle life. Ulcer of the stomach is much more common between the ages of fifteen and

thirty than at other periods, whilst a similar lesion of the duodenum tends to occur later, viz. from thirty-five to forty, if cases following on burns be excluded. Very rarely such ulcers have been seen in both situations in infants, and especially those born in difficult labours, suggesting the probability in these cases of local hæmorrhages into the mucosa and subsequent necrosis resulting therefrom. Half of the cases of acute intussusception are met with in children.

The greater number of cases of carcinoma ventriculi are met with between the ages of fifty and sixty, and very few are seen before thirty, although Dr. N. Moore records a case in a girl aged thirteen, and Drs. Osler and McCrae have collected the accounts of thirteen cases between the ages of ten and twenty. A well-authenticated case of a cylindrical-celled carcinoma in an infant under a month old, probably congenital, was described by Dr. Cullingworth (*Brit. Med. Jour.* 1877, ii. p. 253), and another by Dr. Finlayson (*ib.* 2nd Dec. 1899) in a child aged three and a half years. Sarcoma of the stomach, which is much rarer, has been known in several cases to have occurred under twenty. A similar difference in the age distribution of these two forms of new growth is met with in the intestine, where statistics show that whilst carcinomata occur well after middle life, the sarcomata generally develop before thirty years of age.

Some curious but unexplained differences exist in the *sex* incidence of some diseases of these organs, the most remarkable being in connection with the so-called peptic ulcer, which occurs with more than three times the frequency in young females that it does in males when situated in the stomach, but thrice as often in middle-aged males as in women when located in the duodenum, except as the sequence of burns, when the female appears to be more prone. In the much rarer affection, phlegmonous gastritis, the preponderance in males is very marked, and the same is observed in respect to appendicitis. The frequency of malignant disease of the gastro-intestinal tract is somewhat greater in males, and much more so when the duodenum is the seat of the growth; there is also reason to believe that digestive cancers (including the tongue), which form the greater number of malignant growths in males, as the reproductive organs (including breast) furnish the majority in females, are annually increasing in frequency. Intestinal obstruction due to acute intussusception, volvulus or bands, is more frequent in males, as impaction of gall-stones or of fæces is commoner in women. It is difficult to explain these

facts in any essential proclivity in either sex, and in other affections of the tract no such differences are to be found.

The *state of health* of the individual plays no small share in predisposing to gastro-intestinal complaints; when this is deteriorated the liability to catarrhal and to digestive disturbance is considerable, particularly so in the aged and in the very young.

The predisposing effect of the nervous *temperament* is seen rather in the very important part it takes in determining indigestion, than in the actual causation of structural disease of the organs concerned, though even here its influence cannot be regarded as altogether wanting. The most obstinate disturbances of gastric or intestinal motility or secretion are in many cases solely to be attributed to a neurotic temperament, and persist in spite of the most careful regulation of the other factors concerned in digestion.

Here may be mentioned the peculiar *idiosyncrasies* exhibited by some individuals in respect to certain articles of food, which to the majority are harmless or even beneficial, but to them are positively poisonous. Among such are fish, shell-fish, vinegar, etc. The undue susceptibility of some persons to gastric disturbance from emotional or reflex causes may be referred to.

Race, *occupation*, *season*, *and climate* exert such influence as they do in the causation of structural disease or functional disturbance of the stomach and intestines, less by any direct effect, than by indirectly affecting the general health, the character of the food, and other such circumstances.

Among the immediate or *exciting causes* of diseases of the alimentary canal, by far the most important are errors connected with the *ingesta*. Now the *food* may be regarded as acting as a determinant of gastro-intestinal disease in two ways, separately or combined: either by its direct action on the mucous membrane in virtue of its physical, thermal, chemical, or toxic characters, or when, from its quality or quantity, or from imperfections in the digestive capacity (secretory or motor or both) of the individual, the food is improperly digested, and thereby becomes an irritant to the mucosa, setting up a varying degree of catarrh. Within the former category may be enumerated a diet containing an undue proportion of mechanically irritating particles, such as seeds, fibres, etc.; food that is too hot or too cold, or that is too acid, as vinegar, pickles, or with an excess of pungent condiments; the habitual taking of a large quantity of salt has been known to be provocative of gastric disturbance, whilst a salt-free dietary is also harmful. Or the food

may be actually poisonous from changes that have taken place in it, producing ptomaines and toxic albumoses. The mal-digestion of food and consequent gastric affection may be due primarily to the food itself, or to some digestive imperfection originating apart from the stomach itself, such as a nervous disturbance of its secretory or motor functions. A bulk of food beyond that which the organ can satisfactorily deal with, masses of food inadequately masticated or divided, portions of meat or other nitrogenous substances enveloped in a coating of greasy sauce, an undue amount of sugar, fats, or alcohol, leading to lactic, butyric, and other fermentative changes which give rise to irritating substances, will all tend so to affect the lining membrane of the stomach or bowel as to establish a certain degree of catarrh, temporary though it be, and are much aided by any circumstances which delay the passage onwards of the stomach contents. Obviously, general states of ill-health, such as anæmia, chronic renal disease, nervous exhaustion, mental emotional and bodily fatigue, will, by interfering with the proper secretion and motility of the stomach, bring about indigestion, and thus favour the development of a gastric or intestinal catarrh, if not of other morbid states. Too much fluid at or directly after meals may interfere with digestion by its diluent effect on the gastric juice. Too little fluid may be also detrimental. The taking of food, however good in itself, at too frequent intervals, interfering with the digestion and removal of what is in the stomach, is another cause of dyspepsia, although habit in this as in other cases often diminishes its ill effects.

Most varied are the *toxic substances* that may gain entrance with the food, although they are in no sense a part of it, but merely accidental accompaniments. Among the most important are micro-organisms, many harmless, some pathogenetic. The antiseptic power of the hydrochloric acid of the gastric juice renders by far the larger proportion of them inert, but in the absence of this agent from the stomach, whether from a deficient secretion, or in the intervals when the stomach is empty, such organisms find an opportunity for their development and the production of their injurious effects. In this way persistent suppurative conditions in the mouth and pharynx, carious teeth, and the food and other accumulations which collect under ill-fitting dental plates, etc., may, by the constantly swallowed pyogenic microbes, become a serious and often unsuspected cause of gastric disturbance, by the setting up of a septic catarrh.

Another group is that of the metallic poisons with which the

food may be contaminated, such as lead, arsenic, and copper. The cumulative effect of minute quantities of such substances constantly taken leads sooner or later to gastro-intestinal inflammation. Alcohol, especially apart from food and in the form of spirits, is a potent factor in diseases of the stomach, both acute and chronic, and appears responsible for favouring the incidence of some morbid states by the effect that it has in impairing the digestion as well as by its destructive action on the walls of the viscus.

The injurious results of *wounds*, blows, or severe compression, of articles swallowed, such as pins, bones, etc., or of corrosive fluids, are as obvious as they are seldom met with.

An occasional cause of gastric derangement is a general *adhesion* of the organ, as the result of previous peritonitis, to neighbouring parts. The peristaltic movements may thus be considerably interfered with, and a very painful dyspepsia ensue, leading in turn to a chronic affection of the mucous membrane. Similar effects follow displacements of the organ, and especially abnormal distension of it. These conditions may all be supposed to act chiefly by delaying the adequate removal into the intestine of the stomach contents, and thus favouring fermentations and the formation of irritant substances, the continued effect of which is the establishment of a gastritis. Corresponding lesions of the intestines are liable to bring about serious dyspeptic symptoms dependent on defective motility.

The gastric secretion, and particularly when its *acidity* is increased, as frequently occurs, is itself responsible for certain morbid states by exerting a digestive action on limited areas of the stomach walls, the vitality of which has been impaired by mechanical damage, by degeneration, by obstructed circulation, or by other conditions of disease. It is this which is undoubtedly the most potent factor in the production of gastric ulcer.

Less often, but still with a frequency greater than is probably supposed, the causation of a stomach and probably intestinal affection is to be found in irritating or toxic bodies, such as albumoses, *excreted* by the mucous membrane. Such substances may conceivably have been formed and absorbed lower down in the alimentary canal, and subsequently excreted by the stomach, or they may have originated in perverted tissue metabolism. In this way the gastric symptoms in irregular gout may be explained, though a precipitation of crystalline sodium biurate in the stomach coats has also been held to account for them. The association of duodenal, and less often gastric, ulcer with extensive cutaneous burns has been supposed to be due to poisonous bodies formed at

the seat of injury and eliminated by the bile, causing a duodenitis with ulceration.

The influence of *cold* and *damp* in the causation of gastric and gastro-intestinal catarrh is undoubted, and especially in infants and children, whose alimentary mucous membrane is quite as likely, if not more so, to be affected as their respiratory surface. Individual proclivity in this direction is often to be noticed among adults, with whom changes in the weather are apt to be marked by gastric indigestion, diarrhœa, etc.; and the intensifying effect of cold and wet upon an existing chronic catarrh is well known. The exact pathogenetic bearing of these climatic states is still uncertain, but a disturbance of the circulation, with engorgement of the portal area, seems to be an important factor; whether special poisons are formed by the altered vascularity of skin and viscera is unknown, or whether the congestion simply renders the mucous membranes more susceptible to ever-present injurious influences. *Extreme heat* appears to act by the general exhaustion it tends to produce, rather than by any direct effect upon the stomach or bowels.

Secondary affections of the canal.—There is scarcely any bodily ailment, however slight or however severe, that does not tend sooner or later to interfere with the digestive function, be it only a transient impairment of appetite or lasting state of inflammatory disturbance. The following references to the chief groups of disease will serve to illustrate this statement:—

Indigestion and some degree of catarrh of stomach or bowels is the usual accompaniment of an infective disease, at least during its acute stage, and this has been attributed to an eliminative effort on the part of the gastro-intestinal mucosa to get rid of the toxic substances formed. A family history of tuberculosis has been met with in a considerable proportion of cases of gastric cancer, and in many post-mortem examinations of this disease retrograde tubercle has been found.

As might be expected, an imperfect digestion frequently complicates general states of malnutrition, such as chlorosis and other severe blood affections, rickets, obesity, malignant disease outside the alimentary tract, syphilis, etc. The perverted metabolism which in various ways characterises these maladies affects the structures of the gastro-intestinal canal, and with that it may be supposed the completeness of their functional activity, and hence indigestion is established.

Very marked are the gastro-intestinal derangements that are associated with obstructed portal circulation, whether this be due

to hepatic disease or to morbus cordis, and very obvious digestive improvement will often follow a relief of the congested vessels.

Affections of the canal, secondary to diseases of the respiratory tract, are not infrequent. Vomiting is a common sequence of severe coughing. Tuberculous disease of the alimentary tract has been attributed to the swallowing of sputum from phthisical lungs.

Reference has already been made to the dependence of gastritis, and it may be added of other affections, upon mal-excretion from renal diseases.

The relationship of nervous maladies to functional derangements of the canal is rather to be seen in those cases where pain is a marked feature, or in other affections of the higher brain powers, nervous exhaustion, emotional outbursts, hysteria, neurasthenia, or mental perversions. The influence of these conditions upon appetite and digestion are well established, and the nervous temperament, even within a range consistent with health, has been mentioned as the cause for a large proportion of dyspeptic conditions, presumably by impairing the nerve control of the gastro-intestinal secretion and motility. The violent attacks known as gastric crises which occur in the course of tabes dorsalis claim a more obvious structural change in the nerve centres for their causation. The vomiting in certain cerebral diseases comes within the same category.

Lastly may be mentioned, among the causes of diseases of the stomach and bowels, affections of those organs themselves which stand in a primary relationship to later morbid states. Thus some degree of gastritis is a constant associate of malignant disease of the stomach; dilatation of the viscus is in a large proportion of cases due to obstructive conditions at the plyorus or in the duodenum; a connection has been traced between carcinoma ventriculi and the scar of a former ulcer, although the association is not perhaps so frequent as has been supposed.

Morbid Anatomy and Pathology of the Stomach and Intestines

The usual post-mortem appearances even of the healthy stomach and intestine differ considerably from those seen in the same organs during life. The normal shades of pink which the mucous membrane exhibits according to the condition of digestion, and the delicate velvety look are no longer seen, the surface is pale and grayish in colour, occasionally streaked by the lines of blood-vessels.

or even spotted with minute hæmorrhages, and of a somewhat glairy and viscid appearance. This last is due to a softening of the epithelial cells, which become indistinct in outline, loosened and separated from the basement membrane and mixed with the mucus resulting from the degeneration of the cells. Should death have taken place whilst food is in the stomach and digestion is in progress, the extent of the softening — *gastro-malacia* — is much greater, and may amount to actual destruction of large areas of the mucosa and even of the muscular and serous layer, producing extensive and ragged rents in the organ, with escape of the contents, and similar invasion of the diaphragm and contiguous structures. This post-mortem digestion, which is specially apt to occur in children, is most marked at the fundus, and may only be represented by quite small erosions of the surface. There is usually some, and perhaps considerable, staining of the tissues of the stomach by the blood, which has undergone change by the gastric juice. After a time putrefactive decomposition supervenes. The first part of the duodenum may be involved in the destruction along with the stomach, but on the whole this is rare.

VASCULAR CHANGES

Congestion and hæmorrhage.—As already said, the post-mortem appearance of the gastro-intestinal canal is but little indication of the actual state of its vascularity during life. Under conditions such as portal obstruction or obstructive heart disease, where the due return of blood is interfered with, the resulting congestion of the mucosa may persist to a greater or less extent after death, and an extreme fulness of vessels and coloration of surface may be seen, especially along the curvatures and towards the pylorus. Distinct varicosity of the veins around the cardiac orifice may occur at the junction of the coronary and œsophageal vessels—gastric hæmorrhoids; the non-persistence, however, of such vascular dilatations after death is not to be taken as evidence of their absence during life. It will be readily understood that, where the mechanical congestion has been intense, rupture of the small vessels is liable to take place, especially when these are imperfectly supported, as in the lax tissues of the mucosa and submucosa; hence interstitial hæmorrhages are common in such cases, and according to their size and extent they may reach to the surface, with consequent effusion of blood into the viscus. Grave alterations in the blood, such as leucocythæmia, scurvy, jaundice, or emboli from malignant

endocarditis, may all give rise to hæmorrhages into the mucous membrane and deeper layers. Along with recent petechiæ, or even without them, may be seen spots of pigmentation, the results of former hæmorrhages.

Among other conditions of vascular engorgement of the canal, is an active hyperæmia due to vaso-motor paralysis of the splanchnic area from inhibition of the tonic influence exerted by the sympathetic, induced reflexly or by central (*e.g.* emotional) states. The removal of pressure on the vessels, as of ascitic fluid, by tapping, and, it is said, the suppression of habitual discharges, menstrual and other, will also lead to overfulness of the mesenteric vessels. It is seldom, if ever, that any evidence of these conditions is to be seen after death. Like the normal variations in vascularity which take place in association with the digestive functions—the fluctuating periods of activity and rest—and between which and the commencement of disease no sharp distinction can be made, these states stop short of producing recognisable tissue change and are of transient duration.

INFLAMMATION

The response of the constituent tissues of the stomach and intestines to the infliction of an irritant that exceeds in degree or kind those to which they are normally subject and is yet not of that severity to cause complete destruction—in short, the process of inflammation—varies much with the character of the irritant, and also with the condition of the individual.

Sometimes the entire length of the canal is involved in an inflammatory change, but more frequently the lesion is limited to certain regions. Thus whilst the stomach and intestine throughout may be in a state of catarrh (gastro-intestinal), such as often occurs in infants and children, and also in severe cases of metallic and ptomaine poisoning, the inflammation may be restricted to the stomach (*gastritis*), to the stomach and duodenum, in exceptional cases to the duodenum only (*duodenitis*), to the small intestine generally (*enteritis*, *gastro-enteritis*), to the cæcum (*typhlitis*), to the appendix (*appendicitis*), to the large intestine (*colitis*), or to the rectum (*proctitis*). It is not easy to explain in all cases why special sections of the canal exhibit a proneness to inflammation, though the position of some parts, stomach, cæcum, appendix, and rectum, where the contents are liable to delay in their passage, may be partly responsible. It is seldom, however, that the jejunum is

affected to any extent, and indeed this organ is remarkably free from morbid structural change of any kind.[1]

From the anatomical point of view the inflammation may be catarrhal, corrosive, phlegmonous or suppurative, or membranous, and not infrequently proceeding to ulceration; from the causal aspect it may be simply irritative, toxic, corrosive, or infective, whether pyogenetic or specific (tuberculous, syphilitic, and mycotic); and as regards duration may be acute, subacute, or chronic. Whilst symptoms associated with these different conditions are largely influenced by the particular portion of the canal which is affected, the histological changes which characterise them are very much the same in the different forms whatever the locality involved, owing to the great similarity in structure of the several parts of the tract.

A. *Catarrhal.*—A catarrh (Vol. II. p. 119) of the stomach or bowels is one of the most frequently occurring of all maladies, induced by the numerous errors of diet or improper ingesta to which these organs are exposed. Or it may be that the irritant is to be found in disturbances in the digestive process carried on in these viscera, and that depend upon causes quite apart from structural defects in the organs concerned, these being thus set up secondarily, and in their turn intensifying the existing indigestion.

The mucous membrane in *acute catarrh* has been seen, at least in the stomach, to become of a deeper red from hyperæmia, more marked in places and not completely uniform. In the mildest form the tissue changes pass by insensible gradations from those met with in the course of healthy digestion to a distinct condition of disease. At first the surface is probably drier than normal, but is soon covered with a layer of thick tenacious mucus due to an increased reaction by the epithelial cells, which rapidly undergo cloudy swelling and a more extensive mucoid degeneration than obtains in the normal state. Similar changes, and more especially those of a granular character, are found in the cells of the glands—with the result, in the stomach, of causing a diminution or complete arrest of the pepsin and hydrochloric acid secretion. Moreover, the cells become loosened in their attachments and desquamate, their place, on recovery, being subsequently taken by the round cells which lie

[1] The expressions "dysentery" and "dysenteric" are often applied clinically to severe forms of intestinal inflammation, more particularly of the colon and rectum, and still more frequently to one of the most prominent symptoms thereof, viz. a mucous diarrhœa with the passage of blood, attended with much pain and tenesmus; the writer, however, prefers to restrict these terms to the specific tropical diseases described in Vol. I. p. 402.

among their deeper ends. Meanwhile the lower layers of the mucosa and of the submucosa are œdematous, infiltrated to a varying extent with leucocytes, some of which escape at the surface with the epithelium and mucus. Sometimes the lymphoid follicles in the intestine are enlarged and very distinct, especially in some cases of infantile diarrhœa, and the term *follicular enteritis* has been applied to such. The readiness with which slight hæmorrhages occur in even the milder forms of gastritis and enteritis is a noticeable feature, whether they be into the tissues or an actual escape at the surface. Ordinarily these changes, which are rapidly established, are transient and quickly recovered from, with a complete return of the tissues to their normal state. But should the irritation be frequently repeated or persistent in character, a more chronic change is set up, which is recovered from with difficulty or not at all. There is also a great liability for a catarrhal gastritis or enteritis, however mild, to be attended with some ulceration, or by the presence of superficial erosions, and especially so when the patient's general health is deteriorated.

The characteristic changes associated with a *chronic catarrh* are, in the earlier stages, a proliferation of the columnar epithelium, with or without a similar condition of the true secreting cells, usually associated with an excessive formation of mucus, and an infiltration of the connective tissue with small round cells; in the later stages there is an atrophy of the epithelial and glandular elements of the mucosa, with or without a fibrotic overgrowth, and usually with some, or may be much, pigmentation, causing the mucous membrane to be of a gray and slatey colour with darker spots of variable extent. Apart from the atrophy which a moderate fibrosis entails, the thickness of the membrane increases with the amount of fibrous tissue present. In extreme cases this may be considerable, the surface being raised into irregular projections, sometimes almost polypoid, from the overgrowth in the deeper part of the mucosa and submucosa. In the atrophic marasmus of infants (the "athrepsia" of Parrot) the mucous membrane extensively, though irregularly, denuded of epithelium and with shrunken and wasted gland tubules, which have here and there undergone cystic dilatation, is thinner and smoother than normal and sometimes with but little of the mucous secretion that, in varying amounts, is seen in the healthy state. In the intestine the villi are shrunken and stunted, but the lymphoid follicles may be hard and prominent, or on the contrary atrophied. In the colon the mucosa is more or less covered with a viscid glairy

mucus containing pus, imperfectly formed epithelial cells, and often phosphatic crystals and putrefactive organisms; not infrequently such mucus may be voided in the form of membranous-looking shreds (mucous colitis). This condition may develop as the sequence of repeated attacks of acute catarrh, or may arise with no obvious acute commencement; it is also a constant associate

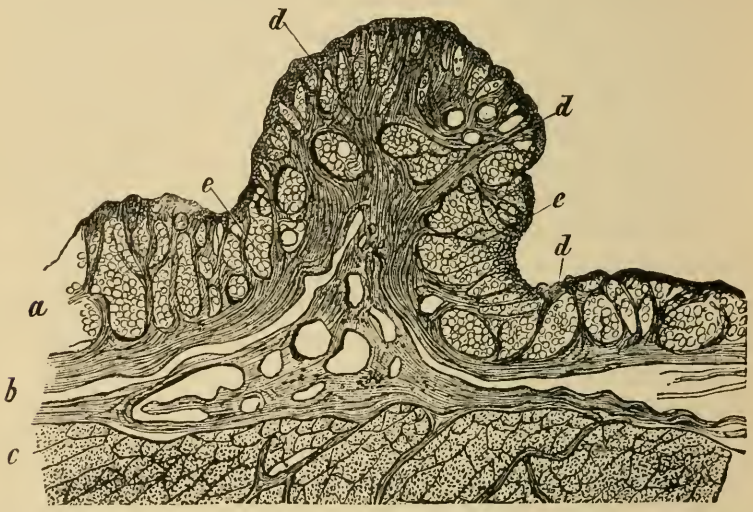

FIG. 4.—Section through atrophic gastric mucosa. *a*, Mucosa with atrophied glands *e*; *b*, submucosa; *c*, muscular coat; *d*, hyperplastic fibrous tissue. (Ziegler.)

of other chronic morbid states of the stomach or bowels, such as dilatation, cancer, etc.; it tends to supervene on a long-continued congestion of the mucosa, as from obstructive heart disease; and it occasionally accompanies disturbances of nutrition, such as chronic Bright's disease, the gastro-intestinal canal possibly taking up an excretory rôle with consequent inflammation from the unaccustomed irritant.

A chronic inflammation of the stomach, and still more of the intestine, that has lasted for long, is most likely to be complicated by some amount of erosion, which may be excessive, at least in adults.

B. *Corrosive.*—When the irritant to which the stomach has been subjected is of an intensely corrosive nature, such as the concentrated mineral acids, strong alkalies, carbolic acid, oxalic acid, zinc chloride, etc., there will follow their ingestion a sloughing of the stomach walls, depending for its extent both in depth and area on the strength and quantity of the poison, and on the presence or not of food in the viscus. The tissues are at once

destroyed, the resulting slough presenting certain recognisable differences according to the destructive agent. Thus whilst the eschars caused by the strong acids are somewhat hard and brittle —mainly from dehydration of the tissues—those from alkalies are softer, moist, even somewhat gelatinous. As regards colour they are generally of a grayish-white appearance, being a dead white from carbolic acid, gray to black from sulphuric acid, and yellow or orange from nitric acid. The tissues immediately adjacent to the necrosed portions are in a state of violent inflammation, extensively infiltrated with blood, which is quickly altered and causes a brown to blackish discoloration, with all the other appearances of acute gastritis to a severe degree. The destruction may involve all the coats and cause perforation of the viscus; this is specially likely to happen with sulphuric acid, but is unknown with hydrochloric acid, and very unusual with the metallic poisons. If any of the corrosive have passed through the pyloric orifice similar appearances will be seen in the duodenum. Should recovery follow, the destroyed areas are replaced by scar tissue which tends to contract, so producing great thickening and perhaps distortion of the organ.

The post-mortem appearances which follow from large doses of the "irritant" poisons, such as the salts of arsenic, antimony, copper, mercury, etc., are sometimes manifested to a greater extent in the intestines than in the stomach, which may altogether escape; the mercurial and antimonial compounds especially affecting the large bowel. Here also the effect on the viscera will largely depend on the quantity and concentration of the poison taken. Areas of intense inflammation of the mucosa, which is covered with tenacious mucus, often blood-stained, will be seen, occasionally proceeding to ulceration or even gangrene, with white, gray, or brownish discoloration, the latter from altered blood; and these appearances may follow the introduction of the poison into the system by other channels than the mouth.

C. *Infective.*—Owing to the inimical effect of the hydrochloric acid of the gastric juice upon most of the micro-organisms that gain entrance to the stomach, gastritis of an infective character is of comparatively rare occurrence. When, however, the acid is deficient in quantity, or the microbe is unduly abundant or very virulent, it may be presumed that inflammation will result from their invasion of the mucosa.

(*a*) The acute exanthemata are usually associated with an inflammatory state of the gastro-intestinal canal, which has been termed *erythematous*. It is not unlikely that this is due to an

elimination of irritant toxins by the mucosa, comparable to what takes place in the kidney. The essential features of inflammation are to be found in varying degrees of severity from a simple catarrh to a membranous gastro-enteritis. The observations of Drs. Perry and Shaw upon morbid conditions of the duodenum led them to notice a close association between enlargement of Brunner's glands and diseases due to specific organisms.

(*b*) *Suppurative or phlegmonous.*—The specific form of inflammation so designated is of infrequent occurrence, and appears in the stomach either as a diffuse or a localised suppuration. In both the submucosa is the main seat of the process, this layer being hyperæmic and infiltrated with leucocytes and a fibrino-serous effusion which commonly permeates among the glands of the mucosa and also invades the muscular layer, and may even extend into the subserous tissue and involve the peritoneal covering, thereby causing a peritonitis either limited or general. In this way the thickness of the stomach walls is much increased and may be dotted here and there with hæmorrhages into the tissues, the glandular epithelium being in a state of cloudy swelling. Such a condition has been compared to an erysipelatous inflammation of the skin. Although several organisms are doubtless responsible for this condition, the only one as yet recognised with certainty is the streptococcus. How the infection invades the stomach wall is often uncertain; as a part of a general pyæmia, the state is intelligible, and wounds, operation and other, of the organ, as well as ulcers or other breaches of continuity of surface may be supposed to permit the entrance of such pyogenic microbes as have escaped the action of the hydrochloric acid, but such lesions are often entirely absent. In the course of the disease the pus may burst through the mucous membrane and discharge into the stomach at many points, giving the thickened boggy walls a honeycombed and broken-down appearance, or the pus may break through the muscular and serous coats into the peritoneum. In the localised or circumscribed variety the collections of pus may form one or more abscesses, ranging in size from a bean to that of an egg (Dr. Leith, *Edinburgh Hospital Reports*, vol. iv. 1896), which may rupture into the stomach, or outwards into the peritoneal or even the pleural cavity or through the abdominal wall.

Occasionally this condition has been known to extend into the duodenum, and still more rarely suppuration may be met with in the coats of the intestine in association with strangulated hernia or intussusception.

(*c*) *Membranous.*—In this variety, which on the whole is extremely rare, the inflammatory products form more or less coherent membranes or pellicles on the mucous surface of a gray, yellowish-gray, brownish, or almost black colour. Their extent is extremely variable, often being limited to the summit of the gastric rugæ or the edges of the valvulæ conniventes or sacculi of the colon, or they may form patches several inches in area, and occasionally a complete cast of the stomach has been vomited, or of several feet of the intestine has been voided. The membranes may be quite superficial, consisting of fibrin enclosing red and white blood-corpuscles, and perhaps a few epithelial cells. They vary from extreme thinness to something under a quarter of an inch in thick, ness. At times the deeper elements of the mucosa are implicated-the necrosis extending down to the muscularis mucosæ; when these membranous sloughs are thrown off or are artificially removed an ulcerated surface is left. The tissues surrounding the patch are likely to be purple and œdematous and tend to become gangrenous. This form of inflammation has been met with most frequently in the colon and the stomach, less often in the small intestine. It would seem that it is oftenest the result of diphtheritic or pneumococcic infection, as shown by the presence of the characteristic organism in the membrane, but it has been found associated with pulmonary tuberculosis, pyæmia, septicæmia, Bright's disease, diabetes, scarlet fever, variola, typhus fever, and has been met with after abdominal injury and hernia; and more than one case is recorded for which no cause could be assigned. Determined as this form of inflammation is by virulent microbes, the membranous sloughs have a close resemblance in appearance and character to those produced by corrosive irritants, especially perchloride of mercury.

It is to be observed that the terms "membranous," or more properly "mucous," colitis, are also applied to a condition characterised by the passage of large quantities of mucus and from time to time membranous shreds and hollow casts of the bowel of even a foot or more in length, not unlike a tapeworm in appearance, and for which they are often mistaken. The basis of these substances is inspissated mucus rather than fibrin, and may contain a few epithelial cells, crystals, and fæcal particles, occasionally giving indications of being formed in successive layers. The inflammatory process leading to this condition is rather of an extreme catarrhal character, and not of the severe type as described above, in which membranous casts of a fibrinous nature are occasionally voided.

Both these varieties of casts are to be distinguished from a portion of the entire mucous membrane which has sloughed away (see *Path. Soc. Tr.* 1897, p. 52).

(*d*) *Tuberculous.*—The stomach very rarely exhibits evidence of tuberculous infection. A mild degree of gastritis not infrequently accompanies an acute tuberculosis or complicates an active pulmonary phthisis, but there is nothing anatomically characteristic in the condition when present. Miliary tuberculosis of the organ has been said to occur. In marked contrast to this, the intestines, more particularly the lower end of the ileum, the cæcum, and the appendix, are often the seat of tuberculous invasion, as part of a general tuberculosis, or limited to those regions. This affection is altogether commoner in children and adolescents. Numerous gray granulations, some quite microscopic, are to be found scattered throughout the submucosa, as well as in the solitary and agminated glands; by coalescence large masses are formed which caseate, break down, and leave characteristic ulcers to be presently described.

(*e*) *Syphilitic.*—Small gummata have been found in the submucosa of both stomach and intestine, occasionally uniting to form large areas in which the mucosa is also involved. Oftener, however, are seen ulcers or cicatrices which have resulted from the gummata, though even these are rare. Some conditions of fibrosis have been attributed to syphilis; and the blood-vessels, in common with those elsewhere, may exhibit the appearance of endarteritis. Ulceration and stricture of the rectum from this cause is of more frequent occurrence.

(*f*) The general characters of *mycotic infection* have been described in Vol. I. p. 244. As involving the gastro-intestinal tract it is found that the more frequent sites of occurrence are the colon, cæcum, and appendix. In the mucosa of these regions firm round or oblong patches are seen presenting yellowish tufts of the fungus and surrounded by a zone of catarrhal inflammation. Such patches are liable to ulcerate, and are often associated with numerous small abscesses in the submucous layer. Mycoses in this situation are extremely prone to be accompanied by pus formation; perforation of the bowel may occur and lead to abscesses which burrow in various directions, especially tending to open externally, sometimes into the bladder. As a further consequence extensive peritoneal adhesions, often involving the abdominal wall, are set up. Mycotic granules have been found in the stools in such cases. Secondary infection of the liver is frequent.

(*g*) Lesions, the result of the invasion of *B. anthracis*, have been

occasionally met with in the stomach and small intestines, and even more rarely in the colon. The mucous membrane is swollen and œdematous, covered by a viscid mucus, and with well-defined patches of sloughing, together with extensive submucous and subserous hæmorrhages. Small ecchymoses may also be found in the swollen mesenteric glands. The characteristic organism is to be found in the affected areas.

EROSIONS OF THE MUCOUS MEMBRANE

Superficial abrasions and destruction of very limited extent—often indeed almost microscopic—may be seen in the course of acute catarrhal inflammation, or as the result of intense congestion or altered blood states—hæmorrhagic erosions—and may in rare cases be regarded as produced by the mechanical injury of coarse particles that have been swallowed, though the solvent power of the gastric juice is doubtless in great measure responsible for those met with in the stomach. Owing to the readiness with which under ordinary circumstances repair takes place in a mucous membrane, such lesions as these are but of short duration and are quickly healed; a slight extension of the process, however, may result in the formation of a distinct ulcer in which the tissue necrosis is more serious.

ULCERATION

Ulceration of the gastro-intestinal mucosa is of frequent occurrence and of considerable clinical importance. The lesion may be single and of strictly limited extent, or large areas may be involved in the destructive change. Essentially a sequel of the inflammatory process and extremely liable to follow on a gastritis or enteritis of whatever character—catarrhal, suppurative, membranous, or specific—it sometimes results from the separation of portions of necrosed tissue that have been caused by other conditions than inflammation, and in the production of which the digestive juices have played no small part. Hence ulceration may be of a primary character, or secondary to some more general condition. Whilst some ulcers run an acute course, tending to healing or to perforation into the peritoneal cavity, others are chronic and progressive, giving but little evidence of repair, and ultimately invading extensive tracts of the bowel, their slow progress allowing of

adhesive inflammation on the serous surface of the gut, thereby averting perforation in a large proportion of cases.

On grounds of convenience only the following grouping of gastro-intestinal ulcers is proposed :—

Inflammatory ulceration :
- Catarrhal, follicular, stercoral.
  - Distension ulcers.
- Following on corrosive poisons.
- Succeeding on suppurative and membranous gastritis or enteritis.
- Associated with chronic perversions of nutrition, *e.g.* Bright's disease ; blood states such as scurvy, leukæmia.
- The result of specific inflammations such as typhoid, cholera, dysentery, anthrax, variola, tubercle, syphilis.

Ulceration of new growths.

Ulceration into the stomach or intestines *ab extra*, as from the gall-bladder.

Ulceration following on the separation of areas of necrosed tissue :
- The so-called "peptic" ulcers of the stomach and duodenum.
- Ulcers consequent on embolism or thrombosis.
- Those resulting from lardaceous degeneration.

Neuro-trophic ulceration.

As might be expected from the very various determining causes, ulcers which are a direct *sequel of an inflammatory condition* exhibit great differences in appearance and character, as also in situation. Taking into consideration all causes, the intestine is oftener the seat of ulceration than the stomach, and the lower portion of the bowel than the upper. As a rule, the more severe the cause of the inflammation the greater is the liability for it to be followed by ulceration, and the deeper and wider will be the destructive process. At the same time the constitutional condition of the patient has to be considered, and where this is deteriorated, as in Bright's disease, gravely altered blood states and other serious perversions of nutrition, the probability of ulceration is proportionately increased.

The simplest form of ulcer, which may follow on any acute or chronic catarrh of the stomach or bowel, appears as a small abrasion of the epithelial layer, which may gradually extend and deepen, until the whole mucosa is involved ; or the first indication

may be a thin glairy pellicle adherent to the mucous membrane, which in time is thrown off, leaving a breach in the subjacent tissue. A very slight loss of epithelial cells facilitates the entrance of micro-organisms or of irritating substances from the canal, thus causing extension of the ulcer. Several small ulcers may coalesce and form a larger one of irregular outline, the edges of which are hyperæmic and swollen, and sometimes undermined, due to the more rapid destruction of the submucosa. A severer degree of this change is represented by the ulceration which succeeds a membranous enteritis, or that follows from the rupture on the surface of purulent collections in the submucosa. To the mildest form the term "catarrhal" is applied, as "follicular" is to those cases where the imflammation and molecular necrosis commence in the lymphoid follicles, which are previously much swollen. Both catarrhal and follicular ulceration frequently co-exist. Such simple ulcers may be met with anywhere in the stomach or intestine, the follicular variety oftener in the colon, and may be single, a few, or very numerous. Under favourable conditions catarrhal ulcers tend to heal completely and without obvious scar, although in the stomach it is possible they may be the starting-point of the more serious "peptic" ulcer. Follicular ulcers of the colon may be extremely numerous, giving to the bowel a honeycombed appearance; they appear in this situation to be very chronic, to show but slight tendency to heal, and rarely proceed to perforation. When the determining cause appears to be scybalous masses in the colon, the ulcers are described as "stercoral," and, as might be expected, are most likely to occur at the more prominent projections in the gut, such as the lips of the ileo-colic valve, the hepatic, splenic, and sigmoid flexures, the rectal valves, and generally the edges of the saccules rather than the intervening surface, and in the cæcum and appendix. It is likely that not only is injury by the hardened fæces responsible for their production, but also irritant substances resulting from fæcal retention and decomposition. A condition of over-distension of the bowel is peculiarly likely to favour the occurrence of ulceration, or to encourage the extension of ulcers that may have originated otherwise. Still more extensive destruction may follow from corrosive and irritant poisons introduced into the canal, proportioned in great measure to the quantity and strength of the material. Healing in such cases is less likely, and when it does take place commonly causes some degree of cicatrisation and consequent deformity.

Infective ulcers.—The ulcers which result from the various forms

of microbic infection exhibit more or less distinctive peculiarities, both as regards their situation and their appearance. Although the organism in some of these cases, *e.g.* typhoid, cholera, tubercle, and anthrax, gains entrance to the body *viâ* the alimentary canal, it is only occasionally that the gastric or intestinal lesion is the result of a direct invasion of the mucous membrane, the inflammation and consequent ulceration generally, though not always, following from bacteria that have reached the gastro-intestinal wall *viâ* the blood current. Some of the ulcers of this group, such as those of typhoid, yellow fever, variola, cholera, anthrax, pneumococcal infection, septicæmia, and erysipelas, run a short course towards healing or perforation; others, such as the tuberculous and syphilitic, are chronic and progressive, though frequently cicatrising in some places as they advance in others.

The special characters of typhoid ulceration, which has been occasionally seen in the duodenum, though most usually in the ileum, cæcum, and first part of the colon, have already been described (Vol. I. p. 90)—those of anthrax (Vol. I. p. 155). The lesions in the other acute infections mentioned are of extreme rarity, and of the character of simple catarrhal ulcers scattered throughout the bowel.

In the same volume, at p. 409, an account is given of the main features of the ulceration that characterises tropical dysentery. Closely resembling this condition in anatomical appearances, but very different in clinical characters, is the malady known as "ulcerative colitis," an unfortunate term, since it is not the only inflammation of the colon associated with ulceration. It occurs with a slight preponderance among females, usually in middle life, but has been seen at the age of twenty. Here also it is the large intestine which is almost, if not entirely, the seat of the mischief, and the entire section of the bowel may be affected, or only parts, or may be a single large patch. In a well-marked case extensive tracts of the mucous membrane are destroyed, leaving the muscular coat exposed, with here and there irregular areas of swollen mucosa with undermined edges. The whole surface looks shaggy and sometimes sloughs may be seen which have not yet separated. A typical case is to be found recorded by the writer in the *Transactions of the Pathological Society of London*, vol. xxxv. 1885, p. 199—the first brought before that Society. The exact etiology and pathology of this affection, as indeed of the various forms of inflammation and ulceration of the colon comprised within the term "dysentery," are still far from known, and until they be so, any scientific arrange-

ment and description of these conditions is impossible. The not infrequent association with Bright's disease is noteworthy. Although it is possible that the disease commences in the lymphoid follicles, as was formerly supposed, there appear to be good reasons for thinking this is not so, and that follicular ulceration, as already referred to, is quite distinct. For apart from the fact that in the cases of ulcerative colitis which have been described no mention is made of the follicles being inflamed or ulcerated in the less advanced areas of the lesion, and that the appearances of true follicular ulceration are very different, there being little or none of those characteristic tracts of destruction and isolated patches of mucosa, and that in short the two forms are never seen to pass into one another, it is difficult to understand why this condition should not more frequently occur in the small intestine or even in the stomach, since the lymphoid follicles are very numerous in those parts. Moreover, as Dr. Hale White has pointed out, "simple ulcerative colitis is usually uncomplicated by any other disease, and in all cases which died it appears to have caused death, but follicular ulceration is usually complicated by other diseases, which generally cause death. Perforation occurs in ulcerative colitis, but not in follicular ulceration."

It has been long known that the inmates of large lunatic asylums, particularly the old and bedridden, irrespective of the form of insanity, are liable to suffer from outbreaks of what is known as "asylum dysentery," which closely agrees in its anatomical features with the "ulcerative colitis" just described. Whether they are really identical is not known. Overcrowding has by some been regarded as an important causal factor, but this is certainly not always the case. Although infection is strongly suggested, no specific organism has been obtained, and it is quite possible that perverted neuro-trophic influence has some share in the production of the malady, or that it depends upon an inflammation set up by the elimination by the bowel of irritant toxic substances derived from the degenerative changes in the nerve centres.

Tuberculous ulceration. — Of extreme rarity in the stomach, tuberculous ulcers are met with with considerable frequency in the intestines, estimates varying from 50 to 90 per cent of all cases dying from tubercle. In 25 per cent of children, the victims of general tuberculosis, the bowel lesion is present.

The occurrence of tuberculous affection of the gastro-intestinal tract is undoubtedly favoured by such conditions as increase the vulnerability of the mucosa, such as chronic catarrh, alcoholism,

and the like, but the disease is determined by the bacillus tuberculosis, concerning the mode of invasion of which information is still wanting. It appears tolerably certain that a perfectly healthy gastric mucosa, with normal acid secretion, would successfully resist direct infection by the bacilli brought in contact with the epithelium, and it is doubtful whether a healthy intestinal mucous membrane would not be similarly resistant. Some observers, however, maintain the contrary. But should the normal condition of the surface be impaired, then the organism has its opportunity, especially, as it would seem, if there should be any breaches of continuity, such as superficial erosions or existing follicular ulcers, with, in the case of the stomach, any diminution in the secretion of hydrochloric acid, which ordinarily is inimical to the development of the bacilli, even if it be not fatal to them. The virus may reach the stomach and bowels in food, meat, milk, etc., derived from tuberculous animals, and this is the usual source in children, thus giving rise to a primary infection of the tract; but even in such cases it is not always easy to exclude a possible derivation from some latent tuberculous focus elsewhere in the body. The invasion of the canal by sputum swallowed from phthisical lungs, or much less often by rupture into the bowel of a caseating tuberculous gland or other organ, are other modes of entry, but it is fair to assume that in such patients the mucosa does not possess the normal resisting power, and so the more readily yields to the organism. The conveyance of the bacillus to the mucous membrane by the blood-vessels is regarded by some as a means of setting up the lesion, if not the only one in the case of the stomach.

The specific tuberculous affection commences in the lymph follicles, solitary and agminated, and the greater abundance of these structures in the lower part of the ileum in great measure accounts for the frequency with which the ulcers are met with in that section of the canal, and their rarity in the stomach and duodenum. The upper part of the colon is very often involved with the ileum, but is sometimes solely affected; a similar restriction has also been noticed in respect to the stomach, where a singular preference for males has been noted. The rectum may also be the seat of the disease. The characters, mode of spread, and course of these ulcers has been described in Vol. I. pp. 225-227. It is to be remembered that simple ulcers may occur in tuberculous subjects, and it is essential that tubercles and the specific bacillus should be found in them to constitute their tuberculous nature. Owing to the adhesions which are formed

at the outer surface as the ulceration proceeds, perforation is commonly averted, though it may happen. Some degree of healing may take place, and in this way cicatricial contraction of the lumen of the bowel may be produced, which is sometimes multiple. A further cause of obstruction is to be found in the adhesions of coils of intestine to each other, or to neighbouring

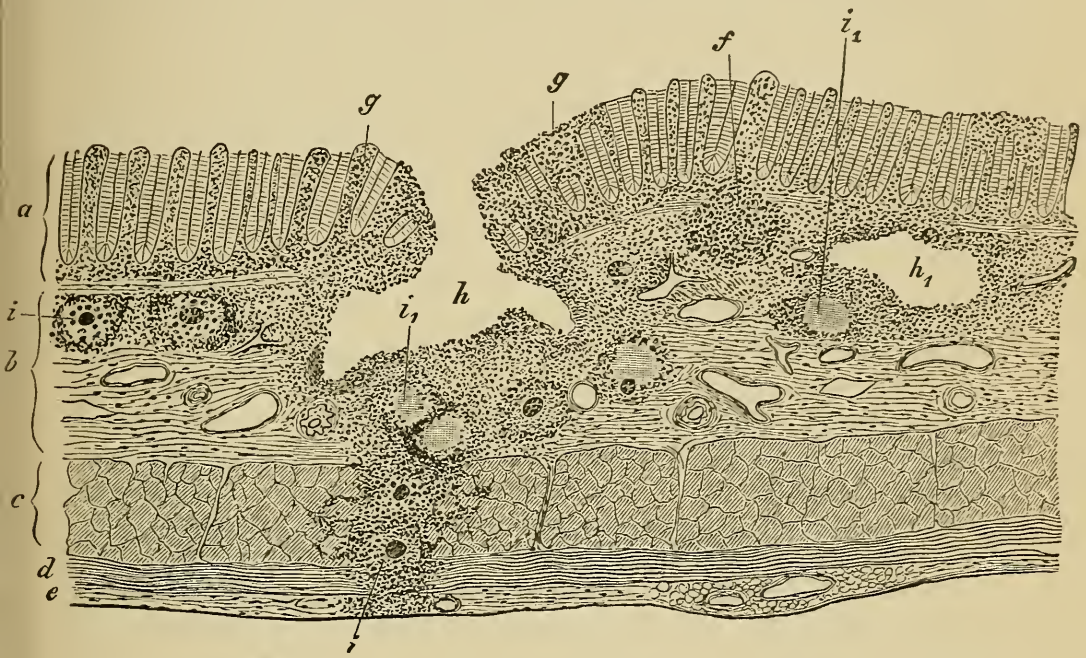

FIG. 5.—Tuberculosis of colon. *a*, mucosa; *b*, submucosa; *c* and *d*, muscular coat; *e*, peritoneum; *f*, solitary follicle; *g*, cellular infiltration; *h*, ulcer; h_1, submucous abscess; *i i*, tubercles. (Ziegler.)

parts due to the coincident tuberculous peritonitis, the virulence of which may be increased by invasion of the serous cavity by the B. coli and other organisms from the gut through the diseased walls. The mesenteric glands usually exhibit some tuberculous change. With the ulceration of the mucosa a lardaceous infiltration may be present, and the occurrence of tubercle in the subjects of syphilis should not be forgotten.

Syphilitic ulceration.—In marked contrast to the frequency with which the mouth, pharynx, anus, and rectum exhibit ulceration in syphilis is the rarity of its occurrence in the stomach and intestines. Nor is it always easy to determine that ulcers found in these situations, though in undoubted subjects of syphilis, are necessarily of specific origin, though the deterioration of the blood in this disease will favour the development of necrotic changes.

The lesions are met with in both the hereditary and acquired forms of the malady, and the majority of the cases—rare as they are—occur in the former, being developed in the later months of fœtal life. They are probably oftenest due to the breaking down of gummata, or to what has been described as a "gummatous infiltration"; and as these morbid conditions originate in the submucosa, the erosion of the mucosa is apt to be of less extent than the destruction of the deeper layer, and consequently the edges of the resulting ulcer are undermined and irregular, the floor being covered by "a yellow tough gelatinous deposit,"[1] thus differing from the clean "peptic" lesion. Miliary gummata are generally to be seen in the adjacent submucosa. Perforation seldom takes place, being prevented by an adhesive peritonitis, characterised by extensive fibrous tissue formation which is set up. Sometimes the ulcer is the result of necrosis of an area of lardaceous infiltration, and syphilitic endarteritis may be the cause in other cases, though the lesion then approximates more to the peptic variety.

Respecting the *ulceration of new growths* it is sufficient to say that the most rapidly growing neoplasms are those most likely to exhibit the greatest degree of destruction, the soft fungoid outgrowths especially so. The solvent action of the gastric and pancreatic secretions considerably aids in the process when the resisting power of the tissue is lowered by being inflamed.

Peptic ulcers.—The stomach and the duodenum—at least as regards its first part—are the seat of a very characteristic ulcerative process which is of frequent occurrence, but with a singular difference in sex incidence; for whilst the gastric lesion is far more common in women, especially young adults, the duodenal affection preponderates in middle-aged males, a fact which suggests that local conditions rather than sexual differences are more concerned in the production of the disease. The duodenal affection has been often met with in children, and even in the new-born, which is much more rarely the case with the gastric. H. Collin (*Étude sur l'ulcère du duodénum*, Paris, 1894), out of 297 cases of duodenal ulcer, recorded 42 in patients under ten years of age. The frequency of this form of ulcer, which is by far the commonest met with in the stomach, has been variously estimated. From the post-mortem appearances either of actual ulcer or of cicatrix, it has been set down as 5 per cent of all autopsies; some observers have stated the percentage to be much higher.

The ulcer or ulcers, for there may be more than one, either in

[1] Hemmeter, *Diseases of the Stomach*, 1901, p. 600.

the stomach only, or both in the stomach and duodenum, present, according to their duration, certain very distinctive appearances. The more recent the ulcer the smaller it is, averaging half to three-quarters of an inch in diameter, of a round or oval shape, with remarkably clean punched outline, with no special thickening of the edges, the adjacent tissues being healthy in appearance except, perhaps, for a few scattered hæmorrhagic spots. The depth to which the ulcer has extended varies, and the base may be formed by the muscular layer or by the peritoneum, or this may have perforated and communication been established with the peritoneal cavity. There is, however, a general disposition for the area of the destruction of the several coats progressively to diminish with the depth to which the necrosis has extended, so that the destruction of the mucosa is greatest, and of the serous membrane the least. In the ulcers of long standing this last character is commonly well marked, so much so as to give a shelving appearance to the area of destruction, the edges of the several layers being more or less distinct; and to this the expression "funnel-shaped" has been usually, if not very helpfully, applied. Moreover, the edges in this case may be, though not always, much thickened from fibroid growth, but not undermined; the size of the ulcer is generally much larger, attaining a measurement of even five or six inches across in extreme cases and of somewhat irregular outline. The chronic inflammation associated with the growth of these slowly progressing ulcers, and that is to be seen in the tissues immediately around the edges, tends to cause adhesions of the stomach to contiguous parts, and this in proportion to the fixity of these structures. Hence perforation into the peritoneum is in some measure avoided, and the floor of the ulcer may be formed by the pancreas, the liver, or even the spleen, far less commonly by the abdominal wall owing to its constant movement, and the ulcer is more likely, therefore, to perforate into the peritoneum when situated on the anterior surface of the stomach. But the same accident may happen whatever the situation of the ulcer, and perforation into the pleural or pericardial cavity, or into the post-peritoneal connective tissue, or the formation of subphrenic abscess, may occur. Or in this way communication between the stomach and the intestine may be established, and even very rarely into the cavities of the heart. Perforations on both surfaces have been met with. Whilst ulcer of the stomach may be found at any part, it is far more frequent in the pyloric region, and on the whole oftener on the posterior wall. Occasionally the ulcer may extend across

the smaller curvature on to the anterior surface. Although the length of time which the ulcer has lasted undoubtedly determines differences in the appearances presented, the writer is unable to recognise that any essential distinction in their nature exists between the two forms here described, and can see no advantage in applying the term "acute" to the former variety, the actual duration of which no data exist for determining, and no clinical justification for drawing such a distinction as the term might imply. Cicatrisation very frequently occurs, and the scar may be seen post-mortem, perhaps in association with a more recent ulcer. Considerable deformity of the organ may result from the shrinking of the scars, especially of those which extend over both surfaces and the lesser curvature, whilst if the pylorus be involved in the cicatrix the orifice may be much constricted.

In offering an explanation for the production of these ulcers, the fact that a healthy mucous membrane successfully withstands the digestive action of the gastric secretions, and that any condition which tends to diminish the resisting power of the tissues favours the necrotic process, must be borne in mind. Indeed, the pathology of gastric ulcer mainly resolves itself into the search for this deteriorating condition, it being fully admitted that the solvent action of the gastric juice is the immediately destructive agent. It is obvious that this action may be at once exerted on a slightly damaged epithelium, but assuming the deeper layers of the mucosa are not implicated in the original damage, whether mechanical, corrosive, inflammatory, degenerative, or other, then the resulting necrosis is limited to the epithelium and a merely superficial erosion is formed. But with a deeper implication a deeper lesion ensues, and ulceration is the consequence. It is probable that the immediate cause is not always the same. The effect of mechanical or chemical damage of the mucosa is obvious, but few cases are to be thus explained, and still fewer by blows or pressure on the epigastrium, though the latter has been put forward in the case of tailors, cobblers, etc., and in tight-lacing. An impairment of the blood-supply by embolism or thrombosis of the small vessels, thereby interfering with the nutrition of circumscribed areas of the gastric wall, is a possible, but certainly not a frequent cause. But it is in this way that minute emboli in pyæmia, or particles that gain an entrance to the circulation as the result of cutaneous burns, may give rise to a few cases of gastric or duodenal ulcer, the gastric juice being the actual destructive agent which exerts its solvent power on the damaged tissues. Endarteritis will also affect the

tissue nutrition, and this may explain the alleged liability to the lesion in syphilis as distinct from the ulcerating gumma that has developed in the submucosa. Dr. S. Martin has described a condition which he has termed "bacterial necrosis" due to the invasion of the mucosa or of the gastric glands by micro-organisms, the tissues being from some cause less resistant than normal. It is not suggested that any special inflammatory change follows on the invasion, comparable to that which leads to the ulceration of the intestine in typhoid or tuberculous infection, but that the tissues invaded become less resistant to the peptic digestion, and necrosis and ulceration in consequence ensue. Whilst it cannot be denied that this is the primary cause of some cases, it can hardly be accepted as other than a very occasional condition. Minute fibro-myomata in the submucosa have also been described by the same observer as a rare cause of ulceration of the superimposed mucous membrane by the pressure which the growths exert and consequent impairment of the vitality of the tissue elements. To the writer it appears for many reasons probable that the most frequent cause of this far from rare lesion is the readiness with which a catarrh of the mucous membrane is induced, and with that superficial erosion just sufficient to give the gastric juice a chance to exercise its digestive action and establish a small area of destruction, which in many cases is prevented from healing by the continued action of the juice and the irritation of food. If, as is often the case, the acidity of the secretion is abnormally increased, its destructive power will be greater. The minute hæmorrhages in the submucosa so frequent in the extreme hyperæmic states to which the stomach is subject, by the pressure they exert and the tissue damage they determine, would also appear to be conditions contributing to the same result. Since also gastric ulcer, at least in its commencement, is most common in chlorotic girls, the general malnutrition in such patients renders the resistant and reparative power of the tissues much less than normal, and consequently favours the production of an ulcer. It has been sought to explain the occurrence of the lesion by ascribing it to a perverted trophic influence exerted through the vagus, but the grounds for such a view are extremely uncertain.

Lesions of identical character to these so-called "round" or "peptic" ulcers are met with in the duodenum, most frequently in the first part, much less often in the second, and most exceptionally in the third, *i.e.* below the entrance of the alkaline bile and pancreatic secretion. A preference is exhibited for the anterior wall. Sometimes there is more than one ulcer, and a similar

gastric ulcer may co-exist; the stomach lesion, however, is much more frequent than the duodenal, in the proportion, it has been estimated, of thirty to one. In origin, appearance, and course they exactly correspond, though perhaps a larger proportion of the duodenal perforate, but healing does take place, and the orifice of the bile-duct may be included in the cicatrix, causing biliary and pancreatic obstruction. The chief point of interest about this form of duodenal ulcer is its association with extensive cutaneous burns, an association too frequent to be mere coincidence, records showing about 6 per cent, but much less frequently than was formerly supposed, probably from the greater antiseptic precautions now observed in the treatment of the wounds. It may be mentioned that severe frost-bite has also been known to be associated with duodenal ulceration. It has been suggested that during the inflammatory stage of the burn septic particles gain entrance to the circulation, and as a result minute hæmorrhagic infarctions have been seen in the mucosa of the alimentary canal, the damaged tissue being then dissolved by the gastric juice. Whilst this hypothesis may satisfactorily account for the absence of such ulcers from other parts of the intestine, it does not explain why the duodenum rather than the stomach is the seat of the ulcer, as undoubtedly is the case. For this reason the explanation given of a duodenitis and ulceration due to excretion in the bile of irritants derived from the burned area may be more valid, except that the site of the lesion is oftener above the entrance of the duct than below.

Neuro-trophic ulcers.—Reference has been already made to the occurrence of a form of ulcerative colitis in the insane; in some spinal cord diseases, such as myelitis and lateral sclerosis, and also in connection with neuritis, ulcers in the intestines have been rarely met with. Provisionally they have been regarded by some as the expression of a perverted trophic influence, similar to the perforating ulcer of the foot in tabes. The lowered tissue resistance under such conditions would favour erosion of the mucosa, and invasion by organisms from the canal, whilst the possible elimination of toxic products of waste of the nerve centres by the bowel might determine an ulcerative inflammation. The explanation of the lesion in some cases of multiple infective neuritis has been said to be a coincident arteritis with occlusion leading to necrosis of tissue and sloughing.

RETROGRESSIVE CHANGES

Atrophy.—A wasting of the tissues of the stomach and intestines occurs as a part of the atrophy of the senile state, or in other cachectic states associated with marasmus. In starvation it is estimated that these organs lose 40 per cent of their weight, becoming extremely thin and transparent. As already described, it is a frequent result of a chronic gastro-intestinal catarrh, though there may be a coincident fibrosis causing a thickening of the walls, whilst the normal tissue elements—epithelial and glandular—have shrunk or disappeared or undergone fatty degeneration. The change is almost confined to the mucosa, the villi especially

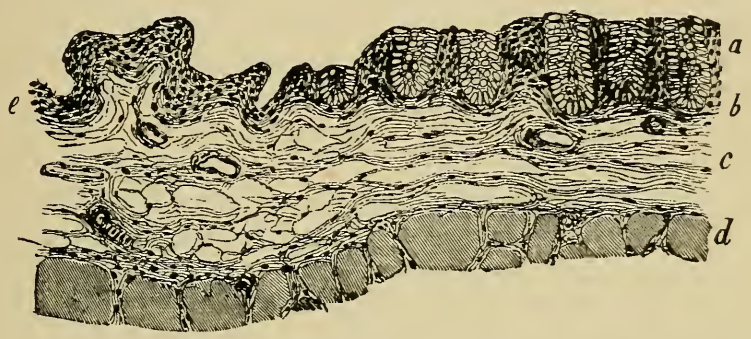

FIG. 6.—Atrophy of colon. *a*, mucosa reduced in thickness; *b*, muscularis mucosæ; *c*, submucosa; *d*, muscular coat; *e*, completely atrophied mucosa. (Ziegler.)

suffering; in extreme cases the thickness of the coat is reduced to a fifth of the normal with complete disappearance of Lieberkühn's follicles. The entire layer is replaced by connective tissue, containing a few round cells, and presents a smooth appearance with irregular thickenings at places; and the muscular tissue and lymphoid follicles are unaffected. Areas of congenitally atrophied muscular tissue have been described by Nothnägel. A similar condition of wasting involving more or less all the coats usually occurs with morbid dilatation of the stomach or bowel.

The stomach is markedly diminished in size in cases of constriction of the cardiac orifice, the walls undergoing a simple atrophy, and also in the condition known as fibrosis presently to be described.

Degenerations.—The inflammatory process involves degeneration of the tissues affected; this as regards the epithelium, columnar and glandular, is *granular* and *mucoid*, whilst the muscular tissue when

implicated presents the appearance known as *cloudy swelling*, with later a *fatty* change. This latter is especially seen in phosphorus poisoning, when the epithelial cells, as well as the muscular layer, may exhibit extreme fatty degeneration. The same may be seen in severe states of anæmia, and it is also the sequence of pressure caused by fibrosis. A few cases have been recorded of extreme fatty degeneration of the ganglion cells of the sympathetic plexuses in the walls of the intestines, associated with marked atrophy of the bowel.

The stomach and intestines may share in widespread *lardaceous* change, particularly in cases of chronic suppuration of pulmonary tuberculosis or of syphilis, which may be almost entirely confined to the blood-vessels of the mucous and submucous layers, sometimes extending into these coats and also the muscular, which become pale and somewhat harder than normal. The lymphoid follicles usually escape the change (see Vol. II. p. 99).

GANGRENE

Owing to the extensive and freely communicating blood-supply to the stomach, gangrene on any extensive scale is seldom if ever seen. A considerable destruction of the walls has been alluded to as the result of corrosive poisons, and sloughing to a limited extent may be seen in connection with new growths, the vessels of which have been blocked, and also in the phlegmonous variety of gastritis. The digestive power of the gastric secretions upon such of the tissues as have lost their proper vascular supply is an important factor in the destructive change, and plays a large part in the production of gastric ulcer. Septic emboli, acute yellow atrophy of the liver, gangrenous stomatitis, pneumonia, and acute tuberculosis, have been noted as causes of necrotic patches in the stomach.

The severer forms of inflammation are likely to lead to gangrene in the intestine also, and in tropical dysentery this may take place over considerable areas, the blackened putrid sloughs being remarkably offensive. Among other causes are thrombosis or embolism of the larger branches of the mesenteric vessels; detachment of the mesentery from the bowel; local constrictions of the gut and interruption of the circulation, as may occur in strangulation of the bowel, or in intussusception, in which several feet of the invaginated intestine have been known to slough away. The appendix, from its peculiar mesenteric arrangement, is singularly

likely to become strangulated and gangrenous. Sloughing even to considerable extent has been known to result from fæcal accumulation.

The gangrene is necessarily of the moist variety. The affected region is at first intensely red, gradually becoming purple and even black, from decomposition and the action of sulphuretted hydrogen on the iron of the effused blood. The tissues also become soddened and swollen, all distinctive characters being lost, and are finally thrown off as a dark gray or black pulpy slough.

FIBROSIS—CIRRHOSIS

These terms denote an excessive overgrowth of fibrous tissue in the walls of the stomach or bowels. In the stomach it is met with chiefly towards the pylorus, or it may involve the entire organ, which in consequence becomes much thicker and of firm, leather-like feel, paler and much less distensible than it should be, and with a capacity diminished to a quarter or less of the normal. In the greater number of cases it is the result of a chronic inflammatory process following on a long-continued catarrh, but it occurs also in cases of corrosive poisoning. This, as already mentioned, is associated with more or less atrophy of the epithelial and glandular elements, and some cystic change in the latter, which is further increased as the new-formed fibrous tissue in the mucosa develops and contracts, compressing the glands and blood-vessels. Owing to an unequal distribution of the morbid change and irregular contractions of the cicatricial tissue, the mucous membrane is thrown into the most varied thickenings, the surface at places assuming a polypoid appearance, constituting the so-called *état mamelonné*. Unless very advanced the change is chiefly restricted to the mucosa and submucosa, but should it extend into the muscular layer, the contractile fibre cells are likely to be atrophied by pressure of the new-formed material, a result which is also to be seen in the muscularis mucosæ. It is to be noted, however, that this fibrotic change is by no means an invariable sequence of a chronic catarrh, even though atrophy of the mucosa should result.

In the somewhat rarer cases when the fibrotic change more uniformly affects the stomach, and to which the term "cirrhosis" is perhaps more usually confined, the association with a chronic catarrh is much less constant, and the condition appears to be rather of the nature of a primary overgrowth in the submucous and subserous connective tissue, the determining cause of which

is seldom obvious, although alcoholism is apparently sometimes responsible, comparable to some cases of cirrhosis in other viscera.

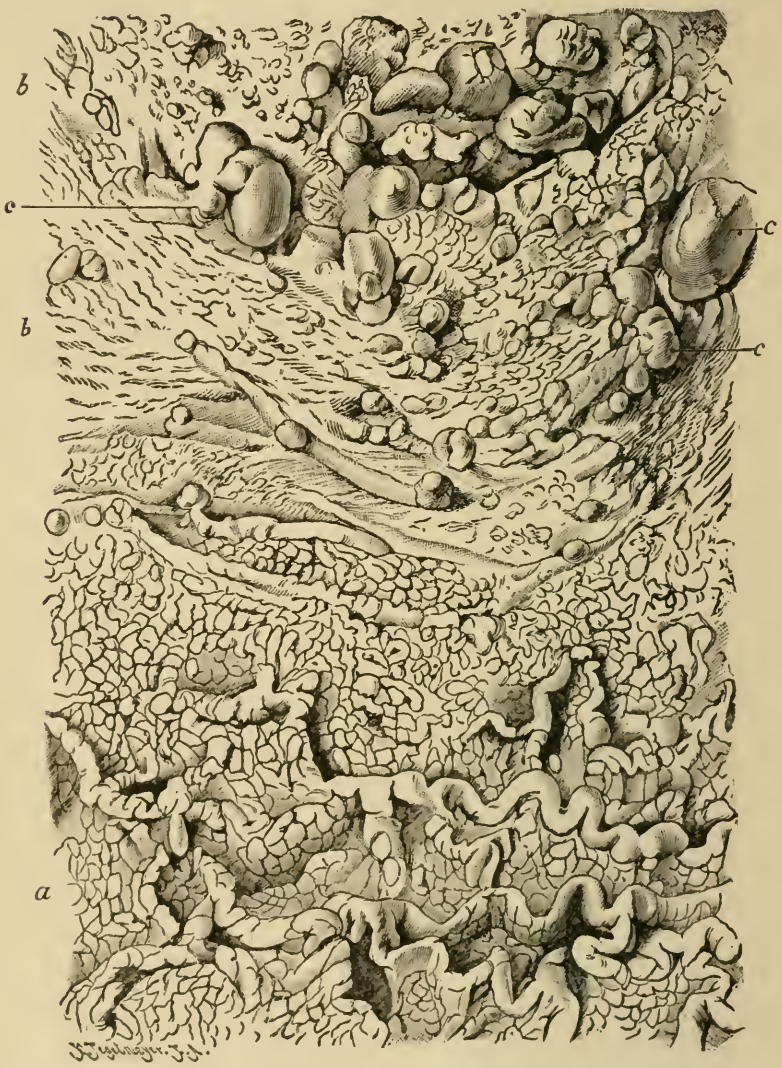

FIG. 7.—Atrophy of gastric mucosa (*état mamelonné*). *a*, normal mucosa; *b*, smooth atrophic mucosa; *c*, polypi. (Ziegler.)

The mucous membrane suffers less both relatively and absolutely than the other coats, and the muscular coat is usually much hypertrophied.

A general fibroid change, also with hypertrophy of the muscular layer, involving sometimes the entire organ, may be associated with

a malignant growth of the stomach, the structural characteristics of which are perhaps best marked or even restricted to the pyloric region, and this is especially likely in scirrhous carcinoma.

Cases are occasionally seen where the fibrous overgrowth is due to an extension from a chronic peritonitis into the subserous and other layers.

An extreme fibroid induration of the pylorus, causing often extreme obstruction, is of not infrequent occurrence, and is by some regarded as essentially a modification of scirrhous cancer, but if this be so, the cases exhibit none of the characteristic clinical features of malignancy.

HYPERTROPHY

An overgrowth of the normal tissues of the canal is seldom to be seen except as affecting the muscular coat, and this only within certain limits. It is most marked in the pyloric region, and is determined by conditions which interfere with the normal passage of the gastric contents through the pyloric orifice; and in a less degree on the proximal side of any persistent intestinal obstruction. When the movements of the stomach are restrained, as by general peritoneal adhesions fixing it to the abdominal wall and elsewhere, or the coils of intestine become adherent to each other from peritonitis, the muscular tissue also tends to hypertrophy. Cases are occasionally seen of very distinct hypertrophy of the muscular coat without any apparent cause.

Attention has lately been called to a hypertrophied condition of the mucosa of the duodenum and upper part of the jejunum in diabetes.

A very rare, but at the same time perfectly well-established affection, is a congenital hypertrophy of the pylorus found in infants a few months old, and leading to death from inanition. The mucosa, and still more the circular muscular ring, is very considerably thickened by an overgrowth of normal tissue, quite distinct from a fibrotic change in the same situation met with at a much later age, though sometimes also spoken of as congenital. Extreme stenosis of the orifice results in some dilatation of the stomach and even of the œsophagus. The cause of this condition is quite unknown.

NEW GROWTHS

By far the greater number of new growths met with in the gastro-intestinal canal are malignant in character, and exclusive of

the uterus, the stomach is the commonest site in the body for the development of cancer.

(*a*) CARCINOMATA.—These are almost always *primary*. Occasionally the *stomach* is involved by the extension into it of a cancer of adjacent parts, *e.g.* gall-bladder or pancreas, or colon, or from the œsophagus.

The sex and age incidence have been referred to in the section on Etiology (p. 148, also see Vol. II. p. 133).

Structurally the tumours consist of a perverted epithelial overgrowth embedded in a fibrous stroma, and upon the differences in the proportion of these elements, the character and mode of arrangement of the cells, the extent of infiltration of the several coats, and the destructive and degenerative changes which may take place in the new formation, depend the differences, naked-eye and microscopic, which these growths present, and to some extent the varied symptoms they give rise to.

Various terms are applied to the several forms, based chiefly upon their gross appearances. Thus scirrhous growths are those in which the fibrous tissue element greatly preponderates, so much so that the cell elements, which in this form are usually spheroidal, may require careful searching for. Like all forms of malignant growth they are most frequent at the pylorus, scarcely if at all extending into the duodenum, and along the lesser curvature, but they also tend to infiltrate the body of the organ, and in extreme cases involve the entire viscus, which is thickened to an extent of half an inch or more, causing an extreme shrinking of the stomach and reduction of its capacity to a few ounces; many cases of cirrhosis ventriculi are doubtless of this nature. The fundus is least often the site of a malignant tumour.

In the medullary variety the growth is much softer, grows more rapidly, and forms extensive polypoid excrescences into the cavity of the stomach. In these the fibrous stroma is at a minimum, and the abundant columnar or transitional cells are clustered in groups or assume a distinct tubular arrangement not unlike that of the gastric glands, although later, as the tubules become crushed and displaced, this arrangement becomes less obvious. These so-called "cylindrical carcinomata" pass by insensible gradations into what are known as "adeno-carcinomata" or "destructive carcinomata"—the term epitheliomata being collectively applied to them. With this form there may also be a very considerable infiltration of the stomach walls beside the massive outgrowths. Owing to their physical characters and rate of development it is the medullary

cancer which is most prone to ulcerate, and also as a consequence to bleed. Portions of the surface may be seen in a sloughing state, and with the continued formation there is a continued destruction of the growth, even to leaving large ulcerating areas with considerable induration and very thickened edges.

The carcinomata that commence at or are confined to the cardiac region are sometimes of the nature of squamous epithelioma,

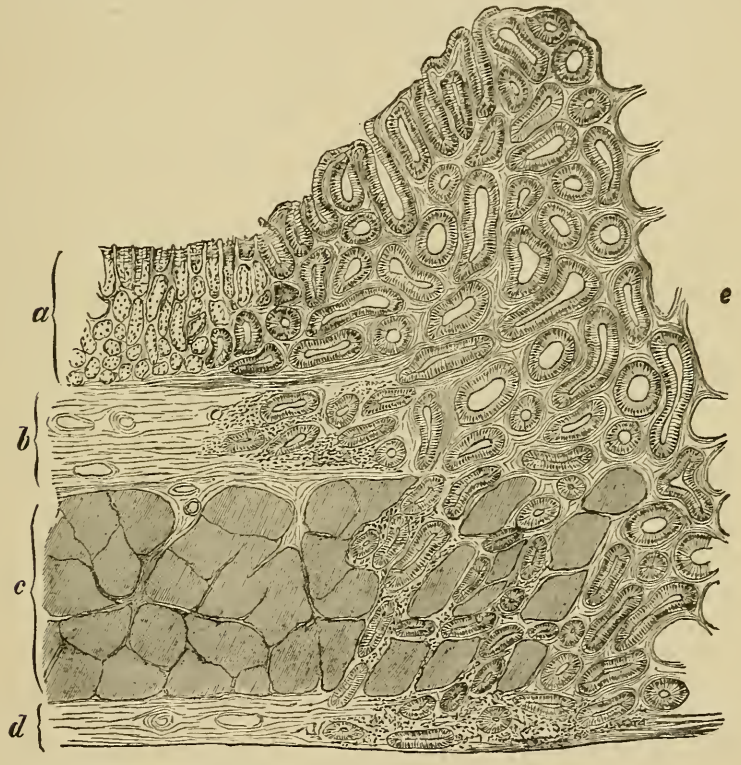

FIG. 8.—Adeno-carcinoma of stomach. *a*, mucosa; *b*, submucosa; *c*, muscular coat; *d*, serous coat; *e*, new growth. (Ziegler.)

such as are met with in the œsophagus, from which they are almost invariably extensions; others are of the spheroidal variety, such as occur in other parts of the stomach. In this condition the stomach generally atrophies and shrinks, the œsophagus being dilated.

These various growths are very liable to undergo a colloid degeneration, both of cells and stroma. To a slight degree it is frequent, but occasionally the change assumes a most extensive character, and gives rise to large alveoli, enclosing gelatinous material, which spread to the peritoneum and thence to the abdominal cavity generally.

Primary gastric carcinoma starts in the mucosa or submucosa, and later invades the muscular, subserous, and serous layers, together with a leucocytic infiltration, especially in the medullary varieties, and a great hypertrophy of such of the muscular fibre cells as have escaped destruction. To what extent a previously deteriorated or damaged state of the constituent epithelial tissues favours its origin is unknown, and in some measure depends on the view taken of the determining cause of the neoplasm, but it is noteworthy that the orifices, especially the pyloric, where the opportunity of friction from the contents is greatest, are the most frequent situations, corresponding in this respect to the growth of cancer elsewhere in the alimentary canal. Some stress has been laid upon the frequency with which a chronic ulcer, or the scar thereof, may become the seat of a malignant growth, but the relationship of these conditions is probably no more than that of a damaged surface favouring the development of the new growth, or on the infective view of the origin of the latter facilitating the invasion of the organism. But in reference to this question the clinical fact that cancer of the stomach very often develops in persons who have given no previous evidence of gastric derangement of any kind must be remembered. When the serous membrane is involved an adhesive peritonitis is set up, sometimes to a considerable extent, the gastric growth being inextricably involved in a mass of fibrous tissue by which the surrounding parts are matted together. This is especially so when the neoplasm has originated outside the stomach, as in the gall-bladder. It may be observed that tuberculous lesions have frequently been found in the bodies of persons dying of cancer of the stomach.

The lymphatic glands along the lesser curvature are involved, and may attain a considerable size: the retro-peritoneal, and occasionally the inguinal, supraclavicular and maxillary glands, may also be enlarged.

Secondary growths are more likely to follow on the medullary forms, which are more vascular, and from which particles are more easily detached into the blood or lymph current. Obviously the liver is the commonest seat of such deposits, and less often the lungs. Subcutaneous nodules, especially around the umbilicus, are occasionally seen. Involvement of neighbouring organs—liver, peritoneum, pancreas, colon, etc.—by extension is common, and in this way fistulous communication of the stomach with the intestines and even through the abdominal wall may be established.

The duodenum, and more particularly the second part, is an occasional seat of primary carcinoma that accords in general characters and course to similar gastric growths. It is by far the most common situation for malignant tumours of the small intestine, and is frequently involved in extensions of growth from the pancreas or pylorus or gall-ducts. It almost always causes stricture of the gut, and, as in the stomach, the scar of an ulcer appears to be not infrequently the starting-point of the new growth. As Dr. Rolleston has pointed out, "carcinoma has a special tendency to arise in the duodenal mucous membrane covering the biliary papilla. This must be differentiated from carcinoma arising inside the papilla or in the ampulla of Vater." And he further adds that the former may be rapidly fatal by causing suppurative inflammation of the bile-ducts.

Primary carcinoma of the jejunum and ileum is excessively rare, especially in the former situation; many of those recorded as occurring in the ileum are situated in the ileo-colic valve. As in other parts of the canal, the growths commence in the mucosa or submucosa and frequently in Lieberkühn's glands, and are usually of the cylindrical epitheliomatous type, which may undergo colloid change; a few spheroidal-celled cases have been recorded.

In the large intestine, especially at the ileo-colic junction, cæcum, and appendix, at the flexures, in the sigmoid and in the rectum primary carcinoma is much more common, and increasingly so in the order named; it has been estimated that rectal cancer forms 80 per cent of all intestinal malignant growths. Here also the neoplasm is usually a cylindrical-celled epithelioma and rarely spheroidal-celled, sometimes forming a tumour involving several inches of the bowel, of the size perhaps of a closed fist, the surface of which is raised into numerous elevations. Oftener it forms much smaller nodules, sometimes polypoid, or plaques, and most commonly occurs as a ring round the gut causing a considerable stricture, from the fibroid induration associated with the cell growth, and altogether of so small a bulk as to give little or no evidence on palpation during life. The growth as elsewhere is prone to ulcerate, and according to its size will obviously more or less obstruct the canal, causing dilatation on the proximal side; the softer and more massive forms may slough to such an extent that the patency of the bowel is but little interfered with. Fistulous communication with the stomach or small intestine, or even externally, may be established by ulceration, or communication with the peritoneal cavity may in like manner be set up. Sigmoid and especially rectal

cancers not infrequently invade the contiguous bones. Colloid degeneration of the growth is frequent.

The colon may be involved by extension from a carcinoma of stomach, liver, or pancreas, and this is frequently the case when the uterus is the primary seat, the rectum being invaded.

In all cases of intestinal malignant disease, as in the stomach, secondary nodules are usually to be met with in the liver, and occasionally in the lungs.

The mesenteric glands are generally affected, though sometimes only very slightly, and the inguinal glands are occasionally enlarged when the sigmoid flexure is the seat of the growth, and thrombosis of the external iliac vein, with œdema of the lower limb, is also liable to complicate a carcinoma in this situation.

Malignant nodules in the stomach, *secondary* to similar growths in the breast or elsewhere, are not unknown; Dr. Hale White says 6 or 7 per cent of all malignant gastric neoplasms are in this category. Secondary deposits are also occasionally seen in the small intestine and colon, commonly on the serous surface.

(*b*) SARCOMATA.—New growths of this type occasionally occur in the stomach and in the intestines, both large and small, presenting in their general features a close resemblance to the carcinomata. They are usually of the round cell variety, and have been noticed especially to invade the muscular coat, spreading from the submucous. Further, it has been noticed that the muscular layer is much less hypertrophied than in carcinoma, owing to its being involved from the outset. The growth of this type appears to cause less contraction of the viscus than carcinoma does, and when the pylorus is the seat it extends more frequently into the duodenum than is the case with scirrhous cancer. As a rule also they occur at a much younger age than does carcinoma, very many being in persons under twenty years old. An interesting report of a case of gastric sarcoma in a child three and a half years old by Dr. Finlayson is to be found in the *Brit. Med. Jl.*, 2nd December 1899; with reference to others, see also a case involving the sigmoid recorded in a child aged three years (*ib.* 3rd June 1899).

(*c*) SMALL FIBROMATA, FIBRO-MYOMATA, AND LIPOMATA, have been recorded as of rare occurrence, and the same may be said for the development of lymphadenomatous nodules as a part of a general lymphoid overgrowth. These growths are generally, though not always, sessile. The pedunculated forms are of a glandular papillomatous character, composed of tubes and spaces lined by columnar epithelium which are embedded in connective tissue of all degrees

of density from mucous (myxoma) to fibrous, and sometimes closely resembling in structural characters malignant growths. These polypi, which vary in size from a pea to a small pear, are found in the lower part of the ileum and more frequently in the rectum, in the former situation sometimes leading to intussusception. Occasionally more than one is present, and sometimes they are very numerous (see *Tr. Path. Soc. London*, 1897, vol. xlviii. p. 83, "Multiple Papilloma of Small Intestine"; *ib.* 1893, vol. xliv. p. 85, "Of Large Intestine").

(*d*) Angiomata.—True vascular tumours are excessively rare, but have been described as occurring in the intestines; and in vol. xxvi. of the *Pathological Society's Transactions* is described by the writer and Dr. Hebb a remarkable case of lymphangiectasis chiefly involving the duodenum and jejunum, in which the vessels after death were engorged with chyle and the lymphatic glands were enlarged from a similar condition, the receptaculum and thoracic duct being normal.

(*e*) Cysts.—Portions of gland tubules which have been occluded may develop into cysts, but they are not of frequent occurrence.

Ziegler refers to an extremely rare appearance, viz. gas-containing cysts occurring in the gastric and intestinal wall or in false membranes due to peritonitis, as well as in the liver and other organs. An investigation of this condition by Dr. Bryant and Mr. Pakes (Guy's Hospital Reports) shows that the gas formation is due to bacilli (especially B. coli and B. aërogenes capsulatus), and probably takes place in the period of death agony when the tissues are imperfectly aërated.

Several instances are on record of congenital dermoid cysts which have been found attached to the rectum or sigmoid flexure, probably originating from the mesenteric canal.

Cystic enlargement of the vermiform appendix or of diverticula of the intestine may follow on closure of their communication with the lumen of the bowel, and accumulation of fluid in their interior—*entero-cystomata.*

MALFORMATIONS AND MALPOSITIONS

These departures from the normal condition may be either congenital or acquired.

(*a*) *Congenital.* — Anomalies of development may be in the direction of deficiency or of excess, and may involve considerable portions of the alimentary canal or only limited parts. As illus-

trating *deficiencies*, complete absence of the stomach has been known, the continuity between œsophagus and intestine being completely interrupted. A stomach congenitally small or with partial separation into two parts by septa has been met with, and the deformity known as the hour-glass contraction appears to be sometimes congenital, and has been regarded as a reversion towards the condition which is normal in some animals. A peculiar condition of congenital hypertrophic stenosis of the pylorus has been already described.

Andral records a case where only a straight tube joined the rectum and œsophagus. The ileum may open upon an ectopic bladder. The rectum may end in a cloaca common to the urino-genital organs, or in either the bladder, urethra, or vagina. The bowel may terminate in a closed extremity anywhere between the brim of the pelvis (the rectum being generally represented by a fibrous chord, though even this may be wanting) and the anal region immediately beneath the skin; the anal pouch, which develops from without inwards, is in the latter case absolutely wanting; and all degrees between this and a pouch that has just failed to establish a junction with the rectum may be met with, producing the lesion known as imperforate anus. The valvulæ conniventes are sometimes wanting, or very imperfect, over varying areas of the small intestine. Congenital constrictions of different parts of the canal are occasionally met with—in the duodenum, either close above the opening of the common bile-duct or at the junction with the jejunum; in the lower end of the ileum, where some abnormality in the closure of the vitelline duct appears to be the cause; or in the sigmoid flexure. Such constrictions may be multiple and of very short extent, the canal being much dilated above, and extremely narrowed and shrunken below. The ileo-colic orifice has been seen contracted to the diameter of a small cedar-pencil. The cause of these lesions is very obscure, though the atresia may be occasionally accounted for by the existence of prominent valve-like folds of the mucous membrane.

Or[1] "the malformation may be of the nature of an *excess*. Thus certain parts of the canal—duodenum, colon, and appendix vermiformis—have very rarely been found double. The commonest of all these malformations is a diverticulum of the ileum, which may arise from the free margin of the bowel from one to three feet above the ileo-colic valve. The cæcal extremity of the process (which is occasionally hammer-shaped) may be connected with the umbilicus by a thin fibrous cord, showing it to be an unobliterated

[1] See footnote on p. 273.

portion of the vitelline duct, and is commonly known as *Meckel's diverticulum.* It varies in length from half an inch to six inches, or even more; its structure is exactly that of the ileum; and it has been found the seat of typhoid ulceration, or of perforation from the irritation of foreign bodies that have become lodged therein. In very exceptional cases diverticula of similar structure have been found protruding from the duodenum, just above the opening of the bile-duct, or from the jejunum; they may even be multiple, but they are not connected with the umbilicus. The vermiform appendix may vary from half to twice the natural size.

"Among *congenital malpositions* may be mentioned complete transposition of the viscera, the cæcum and ascending colon being on the left side, and the stomach, sigmoid flexure, and descending colon on the right, the liver, spleen, etc., sharing in the change. Certain parts only of the intestinal canal may occupy an abnormal situation, as in the various congenital herniæ, of which the location of the stomach in the chest from deficiency or absence of the diaphragm is an example; or the displacement may be due to unusual length of the mesenteries, the cæcum and sigmoid flexure being the parts that present the most usual malpositions from this cause. Thus the cæcum may occupy the left hypochondrium or left iliac fossa, or be found in the pelvic cavity, and other parts of the canal show corresponding changes of place. The sigmoid flexure has been seen lying to the right side of the left kidney, which was situated immediately below the bifurcation of the aorta. Similar displacements are referable to adhesions, determined by intra-uterine peritonitis, which is frequently associated with syphilis."

In the fœtus and at birth the stomach is more vertical than in the adult, and this position may be maintained during life.

(*b*) *Acquired* deformities and displacements of various parts of the canal are very common. Owing to the cicatricial contraction which may follow ulceration the stomach may be considerably distorted, especially when the lesion has affected the walls to any depth, and no adhesions have taken place to adjacent organs. The internal surface may be much puckered by radiating cicatrices, or the organ may be constricted in the middle, causing the "hour-glass stomach," with consequent dilatation or contraction of the two parts into which the cavity is divided. Such a deformity when once established is, of course, permanent, and must be distinguished from a temporary condition, which may be sometimes seen post-mortem, due to a spasmodic contraction of the "pre-pyloric sphincter," corresponding to what normally takes place during

digestion. The shape of the stomach is also much altered in advanced cases of cirrhosis ventriculi, or of general infiltration of the walls by new growth. In such the normal pyriform outline is lost, and the shrunken organ is more tubular in character and much diminished in capacity. The stomach may be also very much reduced in size subsequent to permanent œsophageal obstruction. Stenosis of the pyloric orifice is of frequent occurrence, the various causes of which have been incidentally referred to, and may be collectively enumerated thus: (*a*) congenital; (*b*) fibroid overgrowth, malignant and non-malignant; (*c*) growths in the pyloric region; (*d*) cicatricial contraction of scar following ulceration; (*e*) pressure of growth from without; (*f*) displacement and stenosis of pylorus by adhesions or by dilatation of stomach, causing kinking of the duodenum and functional constriction of the pyloric orifice, the so-called "stenosis by dislocation." As the result of adhesive peritonitis the stomach may become fixed by firm fibrous adhesions to the diaphragm, abdominal wall, spleen, and other structures, and some distortion of the organ may result therefrom. Alterations in shape of portions of the intestine are mainly comprised in the dilatations and contractions which are usually associated with stenosis; the adhesions of peritonitis and abnormal communications established by ulceration may also lead to changes in form.

The stomach and intestines may be displaced from their normal position by outside influences, both when healthy and when altered by disease. Thus the stomach may be dragged on by an omentum which has become fixed by adhesions and even drawn into a hernial sac. It is not often disturbed by tumours, but is likely to be pushed forwards by accumulations of fluid in the lesser omental cavity. Besides the alterations in position assumed by the stomach in consequence of its increase in size, the organ may "drop" from its proper situation as a part of a general ptosis of the abdominal viscera, or may alone be so displaced—gastroptosis. Deformities of the trunk from spinal curvature, tight lacing, etc., may also lead to some change in position of the viscus, but the more frequent cause appears to be an undue laxity, the abdominal walls permitting it to fall downwards and forwards.

The malpositions which the intestines may come to present from changes set up after birth are so variable as scarcely to admit of classification. Herniæ, both external and internal, volvulus, and intussusception are among the well-recognised displacements; but there is scarcely any limit to the changes in position which the traction and pressure of tumours and the effects of peritonitis may produce.

Dilatation of the stomach: gastroectasis.—This is the commonest and clinically most important of the alterations in shape and size to which the stomach is liable. Whilst in a few cases apparently primary in origin, it is far more usually secondary to some definite gastric disease, and of these pyloric or duodenal obstruction, from whatever cause, is the most frequent. The difficulty and delay in forcing the stomach contents through into the duodenum leads to their accumulation within the organ, the muscular coats of which may hypertrophy, particularly in the pyloric region. But this compensatory change is only possible to a limited extent, and should the cause of the stenosis be a carcinomatous infiltration the muscular tissue is displaced by the new growth, and the propulsive efforts proportionately fail. The undue retention of the partially digested food in the stomach soon leads to a catarrhal state of the mucous membrane, which diminishes the general tone and resistance of the stomach walls, and contributes to their yielding, which the bulk of the contents and the gases formed by their fermentation still further assists.

Next in frequency as a cause of gastroectasis is a weakening of the stomach walls, such as follows on a chronic catarrh, and occasionally on an acute attack of the same condition. A too frequent ingestion of food—a habit acquired by many persons—has been held as a cause of gastric dilatation. Should there be, as is likely, from the impaired digestion a state of general malnutrition, or any debilitating disease, such as anæmia, chronic renal disease, etc., the resisting power of the stomach walls will be still further diminished, and so favour the dilatation of the organ. The dilatation in these two groups of cases usually takes some time, weeks or months, to be established, but there is sometimes met with an acute form of this condition, the cause of which is often obscure. To this category belong those cases of gastroectasis, which develop in connection with some of the acute infective diseases, when the walls of the stomach may be supposed to be weakened by the toxic substances produced in the course of these maladies. A paralytic distension of the stomach, sometimes to an enormous extent and involving more or less of the intestines, may occur in acute peritonitis, and may follow abdominal operations. An acute dilatation which sometimes occurs in nervous states, such as hysteria and neurasthenia, or following nervous or bodily exhaustion, is far less easy of explanation. A large secretion of fluid, amounting to several pints, is usually found in these acute cases.

The extent to which the stomach may distend from these

various causes is very variable, occasionally to three or four times the normal capacity. With this extreme enlargement the organ tends to fall in the abdomen, the upper part of which it nearly fills, and the lower margin may reach almost or quite to the pubis, bending upwards from the right iliac fossa towards the duodenum, which remains, except perhaps for the first part, almost constant in position. A considerable depression is often to be noticed at the epigastrium, corresponding to the lesser curvature of the viscus. In other and less extreme cases the stomach lies transversely across the abdomen from left to right hypochondriac region, with the lower limit somewhere below the level of the umbilicus; and in exceptional cases the dilated viscus chiefly occupies the left side of the abdomen, being more vertical in position. These displacements downwards are most marked when the cause is an obstruction at the pylorus, and the greatest distension is associated with non-malignant stenosis. In the enlargement from atony the stomach seems rather to dilate upwards (Broadbent). Except for such thickenings as may be due to new growths, and hypertrophy of the muscular layer, which however is wanting in cases of atonic dilatation, the walls of the dilated stomach are much thinner than normal, though rupture is of doubtful occurrence except there be previous ulceration or injury.

In great eaters, and particularly when the food is chiefly vegetable, the stomach may exceed its normal size and capacity, but so long as there is no undue delay in the emptying of the viscus the condition is not to be clinically regarded as one of disease.

Dilatation of the stomach and displacement of the right kidney have been noted as frequently co-existing.

As the result of obstruction in its lower part the duodenum may become enormously dilated, the stomach being commonly also involved. And this portion of the bowel may also exhibit with the stomach a certain degree of paralytic distension.

The duodenum also appears to exhibit a special proneness to become sacculated as compared with the rest of the canal, and Drs. Perry and Shaw have suggested that this may be due to "an unusual degree of intra-intestinal pressure in this part of the alimentary tube." The base of an ulcer in this situation may yield and give rise to a diverticulum, or the bowel may be dragged on from outside by adhesions, forming "retraction sacculi." Sometimes no such cause is to be discovered. Occasionally two saccules may be met with, usually in the neighbourhood of the biliary

papilla. The pouches are lined by a normal mucous membrane, which is wanting when there has been a previous ulcer, and are formed by all the coats, being about an inch in diameter. Sometimes the sac consists of a protrusion of the mucosa between the separated fibres of the thinned muscular layer, covered by the peritoneum. These "distension diverticula" are also to be frequently seen in the colon, where they may be very numerous, varying in size from a pin's head to a walnut, and may extend into the appendices epiploicæ. In addition to these localised distensions others of a more general character are often observed in the intestines, far more commonly affecting the entire or greater part of the colon and preferably the sigmoid flexure. That the normal bowel is capable of very considerable distension of a temporary character from flatus or even fæces is of common experience, but with the removal of the contents the bowel returns to its normal calibre. Any condition, such as great debility or the febrile state, will favour such distension, and recovery may be longer in being established. In these cases the small intestine may be involved. Chronic obstruction in the course of the canal from any cause is the most obvious cause of this condition, and since such obstructions are much more frequent in the large bowel, it is that section of the tract which is oftener dilated. Among those causes of obstruction fæcal accumulation is probably the commonest, either existing alone or superadded to some structural change which has led to stricture. Hence it is that chronic constipation is very usually associated with a dilated colon to some extent, the two conditions increasing one another. For the fæcal accumulation tends to set up some degree of catarrh from irritation, and this state tends in its turn to favour an atony of the muscular coat, and the resistance and tone of the bowel being thereby diminished, its distension is favoured and the liability of accumulation further increased. Sometimes the catarrhal state may proceed to ulceration (stercoral), and sometimes the muscular coat hypertrophies and overcomes the difficulty for a time, but as the gut distends its power to propel the increasing bulk of contents becomes lessened, and thus contributes to the persistence of the obstruction.

Besides these forms of dilatation of the colon, cases are from time to time met with when the cause is by no means clear, and hence, like corresponding conditions in the stomach, are provisionally termed "idiopathic." Most of the recorded cases have been males, notwithstanding that habitual constipation is more frequent in women. Sometimes the condition appears to be congenital, for

it has been observed soon after birth and in children. A remarkable case of the kind recorded by Drs. Walker and Griffiths (*Brit. Med. Jl.*, 29th July 1893) may be referred to as illustrating the degree of distension that may be attained. A boy in whom the belly was noticed to be unusually large a few weeks after birth, and who was under observation from time to time for the same condition with constipation, died at the age of eleven years. Post-mortem—"the body was emaciated, the lower part of thorax being greatly expanded and thrown upwards. On opening the abdomen, nothing was seen but an enormous coil of intestine, looking like a large leg and thigh, with the knee completely flexed; this was seen to be the transverse and descending colon, which measured twenty-three inches round; the excessive dilatation reached to the sigmoid flexure, the last part of which and the rectum were of normal calibre; there was no appearance of constriction in either rectum or sigmoid." Other cases are recorded in which the distension was as great, or even greater, than this, and in many the sigmoid alone was involved to the extent of filling the whole of the abdomen; sometimes the cæcum shares in the dilatation, the ileo-colic valve becoming practically obliterated. The mucosa is usually atrophied with evidence of chronic inflammation of it and of the submucosa, whilst the muscular layer is more or less hypertrophied. The peritoneal covering has been noticed to be considerably thickened.

RUPTURE—WOUNDS

Rupture of the healthy stomach or intestine may be the result of injury, blows over the epigastrium, especially when distended by a meal, or being run over by a wheel. The existence of ulceration will favour its occurrence. Laceration of the duodenum has also been attributed to severe and prolonged vomiting. The much distended and thinned bowel above a chronic obstruction due to new growth has been known to burst. Perforating and gun-shot wounds of these viscera are of not infrequent occurrence. Rupture of the stomach has also resulted from the careless use of the stomach pump.

Fistulous communications between the stomach or duodenum and adjacent parts may be established by morbid conditions originating in the latter. Thus the gall-bladder or colon may ulcerate through into the duodenum, or abscesses in various situations, hydatid cysts, or aneurysms may burst into the stomach or bowel.

FOREIGN BODIES, ENTOZOA

Various *foreign bodies* that have been swallowed, or that may otherwise have gained entrance into the stomach or intestines, may be found therein. Masses of hair, string, cocoanut fibre, etc., felted together, pins, nails, etc., have been seen in the stomach in lunatics or hysterical patients.

Similar masses, formed of vegetable fibres, husks of seeds, fruit skins, etc., with fæcal particles, may be met with in the intestine, and very rarely hard concretions composed of phosphatic salts with carbonate and oxalate of calcium. These may attain the size of a walnut, and when multiple are apt to be facetted; of a white, yellow, or brownish colour, and showing in section concentric layers. Similar *enteroliths* may be due to excessive ingestion of chalk, magnesia, or salts of bismuth. These bodies are usually found in the cæcum, the appendix, and the saccules of the colon.

After the continued administration of olive oil over a long period, rounded or irregular masses of partially saponified fat may be met with in the intestine, of a yellow or greenish colour, the largest being of the size of a horse-bean. They have a close superficial resemblance to gall-stones.

Biliary calculi may be found in the intestine, having been passed by the common bile-duct, or when very large may have ulcerated through into the bowel from the gall-bladder.

The irritation of these bodies may give rise to inflammation and even ulceration of the mucous membrane, or they may cause obstruction.

Entozoa.—The various intestinal worms which infest the intestines have been described in Vol. II. pp. 5-24, and 36-38.

PIGMENTATION OF THE INTESTINAL MUCOSA

The intestinal mucosa frequently shares in the pigmentation which characterises Addison's disease; this is usually slight, but the writer has recorded a case in which the coloration was excessive (*Path. Soc. Tr.*, vol. xlii. p. 302).

Blackening of the colon, to which region the condition is strictly limited, may result from the deposition of lead, mercury, or bismuth taken by the mouth.

W. H. Allchin.

General Symptomatology of Gastro-Intestinal Diseases

The various morbid structural conditions above described determine the occurrence of certain signs and symptoms, some of which are more or less common to all stomach or bowel affections, as others, whether singly or in combination, are more peculiarly distinctive of individual lesions. But besides these definite diseases of the stomach and intestines, with the manifestations they give rise to, there are many phenomena which are referable to improper digestive changes in the contents of these organs which may have been caused by circumstances quite apart from the viscera concerned, and that are properly those of indigestion or dyspepsia. No doubt primary digestive disturbances that may be set up in the alimentary canal, and not directly due to diseases of the viscera themselves, will sooner or later bring about morbid changes in the organs, and a vicious circle be established; and, on the other hand, primary affections of the gastro-intestinal tissues will, in addition to those symptoms unconnected with digestion, such as hæmatemesis, perforation, etc., also tend, in proportion to their severity and continuance, to set up indigestion. It is not, therefore, sought to attempt any sharp distinction between the clinical phenomena due to the structural lesions and those attributable to the ill-digestion of the contents, but in forming a diagnosis and proposing a treatment it is desirable, so far as possible, to keep these two fundamental sources of symptoms distinct, and to distinguish dyspeptic or functional trouble from actual structural lesion.

INDIGESTION

It is important at the outset to have a clear conception of what is meant by indigestion, a state to which the most varied meaning is attached both by professional writers and by the laity, to the great confusion of the subject. Inasmuch as digestion properly signifies the physico-chemical changes which take place in the food in the alimentary canal, it seems to the writer undesirable and inaccurate to include in its purview the function of the absorption of the digesta, and still more the phenomena of metabolism which concern the destination, changes, and uses of the absorbed material, and for imperfections in which neither the digestive organs nor the process of digestion may be in any way responsible. Hence "indigestion," or its synonym "dyspepsia," is here taken to mean some imperfection in the digestive process, whether it be oral, gastric, or intestinal;

and there is no justification for, or practical utility in, restricting the application of the term to errors of digestion taking place in the stomach only, as is frequently done. Of the four successive stages of the function, the oral or salivary, the gastric or peptic, the pancreatico-biliary and the intestinal, following one after another as they do, it is probable that the gastric is far from being the most important, its deficiency being made up for at other stages (see p. 26).

The almost innumerable circumstances which may lead to perversions of the digestive process—the causes, in short, of indigestion—are capable of many modes of grouping, according to the point of view from which they are regarded. The following is submitted as a convenient plan :—

(i.) *Character of the ingesta.*—In a perfectly healthy organism, working under satisfactory conditions of blood supply and nervous control, the responsibility for a dyspepsia that may be set up will rest with the character of the intake. The nature of a normal dietary has been already described (p. 33), and it remains to consider in what manner this normal may be departed from.

Dietetic errors or improprieties in the ingesta are among the most frequent, as they are the most obvious, causes of dyspepsia. It is not necessary that the food should of itself depart from a physiological standard; perfectly normal articles of diet may be presented under conditions which render them indigestible. Thus there may be far too much taken, or a quantity insufficient to excite the digestive process. The excess or deficiency may extend to all the requisite alimentary principles or be confined to one of them; too much or too little fluid, for example, with meals may each be responsible for an impaired digestion. Or, again, certain ingredients may be so associated as to render them indigestible, whilst taken separately they are open to no objection; such, for instance, are starchy or proteid foods enveloped in fat, as met with in meat smothered in greasy sauces or hot buttered toast or pastry. Ices or iced beverages, perhaps harmless alone, may seriously interfere with the digestion of other substances taken with them. Or food may determine indigestion by being imperfectly cooked, by a too monotonous repetition or by presentation in an unappetising form, thereby preventing rather than inducing the needful flow of digestive juices, with such consequences as that brings about. Probably the most frequently occurring fault is an insufficient subdivision of the solid ingesta by mastication, whether this be due to imperfect teeth, or to a habit of bolting the food. Another and common cause of dyspepsia is irregularity in meals, upsetting thereby the rhythm of

digestion, or, as when too quickly following one another, interfering with the gastric digestion in its progress. Nor must idiosyncrasy in respect to certain articles of diet easily digestible by the great majority be forgotten. These are possible circumstances in which suitable and fit food may become harmful. Often, however, the error consists in some element of the food being distinctly noxious. This condition is represented in its simplest form by articles of diet which contain a large proportion of material which is absolutely indigestible, for the digestion of which, that is, the solvent juices of the canal are incapable. Such are the husks, skins, and seeds of vegetables or fruits and many uncooked food-stuffs. The ill effects of such bodies are mainly caused by the mechanical irritation they give rise to. More serious are the results which follow those ingesta which are more actively irritant or even toxic, such as mineral poisons or the more subtle agents developed in decomposing or tainted nitrogenous food. In estimating the results which follow one or other of these dietetic errors regard must be had to whether it be of occasional or habitual occurrence, and allowance has also to be made for the wonderful degree of tolerance which the human alimentary canal may acquire by habit.

(ii.) A second group of causes is to be found in the various *lesions*, vascular, inflammatory, degenerative, and other, exhibited *in the digestive organs*. It may be affirmed that these diseases act by interfering either with the proper secretion of the several fluids, or with the normal movements of the alimentary canal, or with both. Indeed, it may be said that these two forms of functional disturbance, together with perverted sensations in the tract, underlie the well-nigh countless manifestations of dyspepsia; and it is desirable to realise that these disturbances of function—motor, secretory, and sensory—may result either from primary diseases of the digestive organs, or from morbid states of the central nervous system which may sometimes be induced reflexly.

(iii.) Under the heading, *abnormal conditions under which digestion is performed*, apart, that is, from improper food or primary diseases of the digestive organs, are included a number of morbid states, often remote from the alimentary canal, which have a causal relationship to dyspepsia. Such are—

(*a*) *Temporary disturbances*, whether mental or emotional; muscular and nervous exhaustion. The effect of these conditions in upsetting digestion, destroying the appetite, inducing diarrhœa, nausea, and vomiting is well known.

(*b*) The so-called "*nervous temperament*," a phase of being that

is within the wide limits of health, but is indicative of a constitution unduly susceptible to influences originating both inside and outside the body. A very large number of dyspeptics owe their discomforts to this character of organisation. In such cases the manifestations of disordered digestion, whether motor, secretory, or sensory, are apt to be remarkably uncertain and variable; at times occurring with the most careful diet, at others they are wanting, although there have been marked dietetic errors. Nervous disturbances of other functions usually coexist.

(*c*) The *presence of other diseases* not primarily gastro-intestinal frequently leads to some evidences of indigestion, mild or severe. Foremost among these is the febrile state, which almost invariably causes an impaired appetite, thirst, and a tendency to constipation, and not infrequently nausea and vomiting; the digestive powers of the saliva and gastric juice in fever are also known to be deteriorated, and it is probably the same with the pancreatic and other secretions. A like result is very apt to follow sooner or later on more chronic disturbances of nutrition, such as malignant disease of organs other than the alimentary, diabetes mellitus, gout, and the like. Toxic bodies of autogenetic origin appear in some cases to be eliminated by the gastro-intestinal mucosa, and the same occurs with some poisons, *e.g.* morphine, of extrinsic source, and it has been here suggested that this perversion of the excretory functions of the canal may act unfavourably upon the digestive changes going on therein.

(*d*) Another condition which interferes with the proper digestion of the food is an *imperfect removal of the already digested material*, whether by absorption into the blood-vessels and lacteals, or in the stomach, by undue delay in the propulsion of the chyme through the pylorus. This latter may be the result of gastric atony or stricture at the orifice, as the former depends upon structural changes in the mucosa, such as atrophy, or upon an obstruction in the circulation producing congestion.

(*e*) *Abnormal bacterial fermentations* in the stomach and intestines are undoubtedly responsible for many dyspeptic symptoms. How far the normal micro-organisms of the canal may, under altered circumstances, become pathogenetic is, as previously said, unknown, but it is certain that under abnormal conditions of digestion harmful microbic action may be set up, as, for example, lactic and butyric acid fermentations in the stomach, in the absence of a proper quantity of hydrochloric acid. In this connection may be mentioned the passage of pyogenic, tuberculous, and other organisms from the

mouth or pharynx, which may bring about their ill effect lower down in the canal.

Although these several groups of causes are here set forth *seriatim*, it is seldom one acts alone; the establishment of one sets up others, with the result of producing a complex that may offer the greatest difficulty in unravelling. Nevertheless this must be attempted, for the success of treatment in great measure depends on an accurate recognition of the cause.

The **signs** and **symptoms** of structural diseases of the stomach and intestines, together with those referable to mal-digestion of their contents, whether there be obvious lesions of these organs or not, may be conveniently grouped into (*a*) local and (*b*) remote. With respect to the former it is certain that they are present in a greater or less degree whatever may be the character of the cause, and in the more pronounced and graver structural affections they may almost be regarded as paramount, the remoter manifestations being in the greater number of cases scarcely existent. On the other hand, many dyspeptics, in whom the condition is chiefly functional, with little if any recognisable change in the stomach, manifest symptoms which are in a great measure distant from the organs in which the faulty process is at work. At the same time it is not suggested that it is possible to draw therefrom a symptomatic distinction between the organic and the properly functional diseases.

I. The LOCAL SYMPTOMS of gastric or intestinal disorder are separable into (i.) subjective sensations—sensory neuroses—and (ii.) objective phenomena.

(i.) *SUBJECTIVE SENSATIONS.*—The process of digestion is normally carried on without any conscious sensation thereof, and the only sensory impressions connected with the function are a feeling of hunger referable to the stomach, which may in some cases become painful in its intensity, and the sense of pleasurable well-being which follows on the healthy gratification of the *appetite*. This last, however, is rather of the nature of a general sensation than specially associated with the stomach; similarly the sensation of thirst is of a general character, probably connected with a want of fluid by the tissues, and though perhaps most felt in the pharynx, really originates in the nervous centres. Reference has been made (p. 22) to the great importance of a healthy appetite as an excitant of the gastric and salivary secretions. Although essentially of central origin, it nevertheless appears that the appetite might be reflexly excited or encouraged by afferent impressions from the gastric mucosa. Perversions of these sensations—hunger and thirst—

whilst among the commonest symptoms of disordered digestion, and also in some measure of structural diseases of the stomach, are very frequently the expression of central nervous states.

Anorexia or loss of appetite occurs in all degrees from an absolute loathing for food to a slight disinclination, or that modification of a diminished appetite which consists in the quick satisfaction of what is apparently a normal or even increased desire for food by a very small quantity. The condition appears to be closely connected with a diminished gastric secretion, and hence is present more or less in all forms of gastritis, acute or chronic, and since these states complicate very many diseases, especially those of a febrile character, an impaired appetite is one of the most frequently occurring symptoms. Those morbid changes which lead to the destruction of the gastric glands, such as atrophy of the mucosa or malignant disease, are invariably accompanied by a progressive loss of appetite. Excitement, anxiety, or depression are likely to lead to anorexia, often with nausea; and the same may result from drugs, such as alcohol, opium, tea, tobacco, or cocaine; both hysteria and neurasthenia are also causes. An inability to take food on account of the pain it produces, as is usual in ulcer of the stomach, must not be mistaken for anorexia.

The condition known as "anorexia nervosa," when a most extreme emaciation takes place, often with complete loss of appetite, is not to be regarded as merely the result of a want of food or a peculiar form of gastric neurosis, but rather as some abnormal state of the central nervous system, which leads not only to impaired appetite (not always, it is to be noted, a complete anorexia), but also to a grave deterioration in the nutritive capacity of the tissues (see Vol. III. p. 374).

An inordinate craving for food—*bulimia*—appears to be less associated with gastric conditions than with general bodily states. Thus it is well marked in diabetes, and is frequently experienced after acute illnesses, such as typhoid fever or pneumonia, when the tissue waste has been considerable. When also gastric digestion is unusually rapid, and the stomach becomes more quickly empty, an increased appetite is noticed, and this is very likely to occur at night after the last meal. Some forms of intestinal irritation, as from worms, or even from catarrh, appear sometimes to be able to induce the condition, as well as such remoter states as hysteria, and it is occasionally associated with gastralgia and hyperchlorhydria. Mr. S. Paget (*Tr. Clin. Soc. Lond.* 1897) has collected a remarkable series of cases of cerebral injury or disease, in which a voracious

hunger or thirst, or both states, developed, and after lasting for weeks or months disappeared. They appear to have been quite independent of diabetes. Sir William Gowers had previously recorded the association of bulimia with brain tumour, and the same has been reported in connection with affections of the vagus nerve.

Disordered appetite or *pica*, leading to the taking of such things as paper, chalk, slate pencil, string, and the like, has been observed in pregnant women, and also in hysteria, and still more frequently in idiots and lunatics. In these cases it is the central nervous system rather than the stomach which is responsible. Among children, and to a much less degree among adults, a dislike for perfectly wholesome articles of food may exist, and this to such an extent as to render their dietary extremely and undesirably limited.

An excessive *thirst* generally characterises an acute gastritis, and, as a rule, where there has been much vomiting. Where the tissues have been drained of fluid by profuse perspirations, by the passing of large quantities of urine, by hæmorrhage, or by excessive diarrhœa, as in cholera, thirst is complained of, and it is a constant symptom of the febrile state.

The strictly local sensory affections accompanying indigestion and gastro-intestinal diseases exhibit the widest range of degree and character. The channels by which they are probably conveyed to the sensorium have been mentioned in the section treating of the anatomy of these organs. The generally accepted terms in which these sensations are conventionally described are for the most part very inadequate, but the limitations of language in this respect offer no better alternatives. Vague discomfort referred to the gastric region, a feeling of general abdominal uneasiness, a sense of "weight," "fulness," "tightness," or "oppression," are some among those most frequently complained of, and are oftenest attributable to a larger bulk of contents, whether solid, liquid, or gaseous, than the stomach can cope with, whether this has been due to a larger meal than was suitable to the individual, or to the development of gases during the process of digestion, or to the presence of some specially indigestible article of food. Speaking generally, such symptoms are an expression of a delayed digestion, whether from insufficiency of the gastric juice, or from impaired motility of the organ, as in gastroectasis or atony from whatever cause, or from both. A "sinking" sensation before or after food referred to the upper region of the abdomen is among the most characteristic. Prof. Allbutt describes it as a feeling of "utter exhaustion; the voice becomes weak and slow, and the face turns gray; but the pulse does not falter, nor does the

attack feel like a faint, or lead to it." A feeling of "emptiness" akin to hunger is, as the word implies, commonly due to a want of food in the stomach, which may be the result of a very rapid digestion and expulsion of the digesta into the intestine, coming on unduly soon after a meal. The nature of all these sensations and of pain, next to be considered, is clearly related to the sensibility of the mucosa. Normally this appears to be but slight in the stomach; ordinarily, hot or pungent substances swallowed produce no conscious effect when they reach the stomach, and moderate irritation of the gastric mucous membrane seems to cause a sensation of "weight" rather than pain (Brunton). The mucosa, however, is liable to become hyperæsthetic, and then pain is experienced, or sometimes a burning sensation which is almost peculiar to "nervous dyspepsia."

Pain in the stomach, *stomach-ache*, or *gastralgia* is the most marked, the most constant, and in some respects the most distinctive of gastric symptoms, indicating (*a*) some structural change in the organ, such as an ulcer or cancer; (*b*) an irritation due to some digestive disturbance, whether this be an irritant element of the food or a hyperacidity of the secretion, or an acid formed during digestion, such as butyric, which the late Sir William Roberts regarded as more acrid than lactic, or even hydrochloric; or (*c*) some purely neuropathic condition—neuralgia.

The epigastrium and lower end of the sternum are the most frequent situations of pain of stomach causation, being occasionally strictly circumscribed, and perhaps felt in an area that may be covered by the finger tip, in other cases it is more diffused over the epigastric and hypochondriac regions. Less often than in intestinal and other abdominal affections it is experienced about the umbilicus. Owing to the course of the afferent nerve channels, the pain is frequently referred to the interscapular region and to the shoulder, whilst the overlapping of the pulmonary and gastric nerve-supply accounts for the occasional reference to the stomach of pain that is excited by diseased states of the lungs or pleuræ.

In character the pain varies from a dull ache or sense of weight and tightness, to being "throbbing," "spasmodic," "dragging," "gnawing," "darting," "lancinating," "smarting," "burning," or a sense of "soreness," of all degrees of intensity, from simple uneasiness up to intolerable agony. The milder forms may become "wearing" when long continued. It is noticeable that patients rarely if ever use, to describe their suffering, such similes as being "gnawed by a rat," or that their "insides are being burned with

red-hot irons," as they not infrequently do when the lower intestines are the source of the pain, and that gastric pain, whatever be its character, is less likely to provoke mental delusions than chronic intestinal distress is. The duration of the discomfort may be extremely brief or almost continuous. It is seldom that the pain has any reference to the mere quantity of the ingesta, but the quality of the same is apt seriously to influence it, and the simplest articles of diet may in some circumstances provoke great distress. Curious idiosyncrasies in this respect are to be met with. The time relation of the pain to the taking of food, whether it follow immediately, or not for half an hour, or for one or two hours, and to what extent it is relieved by vomiting or by the eructation of gases, or whether it be increased or diminished by pressure over the stomach, are also characters to be noted, as well as the presence or absence of tenderness in the painful area, which is sometimes so great as to prevent contact with the clothes. To some extent these several characters of gastric pain are distinctive of the various diseased states which the stomach may exhibit, and so far are referred to in treating of the special symptomatology of these maladies; and although taken with other indications are of considerable diagnostic value, yet require judgment for their interpretation owing to the exceptions which must be made in any general statement concerning them. It is also important to remember that pain in the situation and with the characters here described may be due to causes other than gastric, such as affections of the colon, spleen, gall-bladder and ducts, spinal caries, thoracic aneurysm, intercostal neuralgia, pancreatitis, appendicitis, or floating kidney. When pain is the most prominent feature, and is unaccompanied by other gastro-intestinal symptoms, especially if it persist, then a suspicion as to its stomach origin should arise, and the likelihood of its being due to some other cause, such as spinal caries, be considered (Treves).

"In cases where pain is present after the ingestion of food, as in gastric ulcer, the higher the area of pain and tenderness, the sooner after food will pain arise. Thus in those cases where the seventh dorsal segment is necessarily affected, and the maximum pain and tenderness is situated at and around the ensiform cartilage, the pain will come on within half an hour of taking food. On the other hand, when the ninth or tenth dorsal is mainly affected the onset of pain may be deferred for an hour and an half. Thus it would seem that the cardiac end of the stomach stands in relation mainly with the sixth and seventh dorsal, the pyloric end mainly with the ninth and tenth dorsal areas" (Dr. Head, Quain's *Dictionary of Medicine*, 3rd edit. 1902).

The sensory neurosis known as gastralgia or gastrodynia, although unconnected with any as yet discovered structural abnormality, is none the less a very real clinical entity, and to be regarded as a neuralgia of the gastric mucosa, characterised by pain referred to the stomach, independent of such gross lesions as ulceration, and independent of hyperacidity. It may, and frequently does, however, coexist with these conditions, and contributes to the pain for which they are then but partly responsible. It is pre-eminently a manifestation of the neurotic temperament, and occurs chiefly in young women, though by no means confined to them. Other evidences of a nervous constitution usually coexist, or a history of former neuralgias, of hysteria or neurasthenia, is generally to be obtained, all of which has an important bearing on the diagnosis of the malady. Although frequently developing without any obvious exciting cause, gastralgic attacks may be traced to fatigue or exhaustion of whatever kind, grief or anxiety, to improper feeding, and still more often to menstruation, occurring in those constitutionally predisposed. The gastric crises of tabes and other spinal cord diseases may be regarded as severe examples of this condition, with a definite nervous disease underlying it. An unexplained but apparently definite association with aortic regurgitation has been pointed out, frequently as a terminal phenomenon with vomiting, in death from that lesion.

The pain tends to exhibit a paroxysmal character, with intervals of varying length, and sometimes with a periodic feature. It appears in all degrees of severity and variety of character, imperfectly described by the terms already mentioned. The milder manifestations pass insensibly from the sensation of "sinking" above described to very definite pain, lasting but a short time or for several hours. Attacks of gastralgia, however, may be alarming in their intensity, and even fatal, the patient rapidly falling into a state of collapse, with cold sweats and low tremulous voice, and, as the writer has occasionally observed, with cramps in the limbs; but the pulse, though small, and either frequent or infrequent, possesses a fair amount of tension that does not accord with the general condition, and may give a clue to the real nature of the case. The pain is, on the whole, less localised than that of ulcer or carcinoma, and may be relieved by pressure, and though it may radiate over the abdomen, is less prone to be felt between the shoulders or in the back. A diffuse tenderness may also be noticed. A copious flow of pale urine, as with other forms of "nerve storms," may follow the attack. Many of these more severe cases closely resemble spurious angina pectoris.

The relation of the pain to the taking of food is a point of importance. In a large number of cases it is felt quite independently, the attacks coming on when the stomach is empty, and not infrequently in the night or early morning; food in such cases may give relief. In both these respects the affection resembles the pain due to hyperacidity. But in a large proportion of cases a meal may set up pain, and this may continue until it is relieved by vomiting, thus simulating a gastric ulcer, from which, in the absence of hæmatemesis, it may be impossible to distinguish it. A consideration of the history and associated symptoms may serve to make a diagnosis, though the not uncommon association of gastralgia with gross lesions must not be forgotten. If the malady last any considerable time the patient may waste from the insufficient food taken during the attacks of pain, but in the intervals the general nutrition is restored; and the appetite, even when there is no discomfort, is often capricious, rendering the dietetic treatment generally unsatisfactory.

From this it will be seen that it is often extremely difficult entirely to exclude the existence of any lesion in a case of gastric pain, and to be sure that it is a gastrodynia only. Attention should be paid to the presence of other hysterical or neurasthenic symptoms, and the relation of the pain to other evidences of disordered stomach, such as vomiting, cardialgia, flatulence, tenderness, etc., as well as to the results of physical examination of the abdomen. The occurrence of pain independently of food, or its relief by the same or by pressure, and a general tendency of periodicity in occurrence, often regularly at night, and but little disposition to be referred to the interscapular region or shoulder, are of considerable importance in arriving at a diagnosis.

Colic,[1] *enteralgia*, *belly-ache*.—In normal conditions the functions of the intestines are performed without giving rise to any conscious sensation; nor does excitation of the mucosa by mechanical, thermal, or chemical irritants ordinarily cause pain. Yet certain dietetic errors, particularly iced drinks, and some drugs, such as arsenic, and most of the vegetable aperients, will cause pain, the latter probably only after being absorbed. Stimulation of the central end of a divided splanchnic nerve produces pain, doubtless due to the myelinated fibres of the afferent spinal roots. As compared with the stomach, the intestines are certainly less liable to pain, and the difference on this point in respect to ulceration in the two regions is remarkable. Nevertheless, certain struc-

[1] This term, originally designating pain in the colon, is used in reference to similar paroxysmal pains in other organs, renal, hepatic, etc.

tural lesions of the bowel, especially acute enteritis, appendicitis, conditions leading to obstruction, whether such as hernia, volvulus, etc., or by external bands constricting the gut, blockage of the lumen by fæces, gall-stones, etc, and new-growths, are frequently, and indeed usually, associated with more or less pain. Whether any lesion limited to the mucous membrane is capable of exciting painful impressions is doubtful; but should the peritoneum be involved the liability will be increased; and, speaking generally, the affections of the colon are somewhat more prone to give rise to pain than diseases of the small intestine. The commonest form of intestinal pain is termed "colic," in which the idea of a painful muscular spasm is involved; this spasm is local, affecting only a portion of the bowel at a time, but there are differences of opinion as to whether the pain is a consequence of the muscular contraction, or whether they are coincident phenomena of a combined sensori-motor character. Certainly exaggerated peristalsis may occur without pain, but whether tetanic spasms may is uncertain, though it is held by some as possible, just as pain may apparently be present without any disturbance of motility. Certain forms of toxæmia, particularly plumbism (Vol. II. p. 65), and in a less degree gout, frequently cause colic, and often of a severe type; structural changes in the nerves have been described by some in cases of lead poisoning. Extreme flatulent distension of the bowels is also accompanied by colic. And among rarer causes may be mentioned purpura hæmorrhagica, due probably to bleeding into the wall of the gut, and angio-neurotic œdema. There appears to be good reason for believing in the existence of a true neuralgia of the intestinal nerves from the mesenteric plexus—an enteralgia—in which no lesion of the bowel or its nerves is discoverable. The occurrence of abdominal pain, apparently of intestinal origin, is a well-recognised symptom arising in the course of hysteria, neurasthenia, hypochondriasis, and sometimes also in such definite maladies as tabes dorsalis. Or the discomfort may be reflexly induced from affections of other abdominal viscera. Exposure to cold or chill is another condition not infrequently followed by pain in the bowels, and this at present can only be regarded as neuralgic in character. Very considerable pain is associated with the discharge of large quantities of mucus and mucous casts from the colon. There is some reason to consider that this form of colitis is largely nervous in character, a hypersecretion of mucus comparable to secretory neuroses elsewhere.

Intestinal pain, from whatever cause, is extremely variable in

degree and in character. Most often it is described by the term "griping," but such expressions as "cutting," "tearing," and "boring" are frequently applicative, and sometimes it is "dull" or "wearing." It is usually paroxysmal, lasting for a few minutes or longer, and recurring at intervals of a few minutes, hours, or days, dependent on the cause. Most commonly the discomfort is referred to the umbilicus, from which it may radiate in various directions. Pressure over the lower part of the abdomen generally gives relief, and the patient doubles up his body and draws up the legs as the attack comes on. When the affection is of an hysterical or neurasthenic nature, some tenderness over the abdomen may be present.

Associated with the pain are numerous other symptoms, those which are attributable to the underlying cause, and also others, for which the pain itself may be looked upon as determining. In the mildest cases nothing beyond transient colicky pains—tormina—possibly with some intestinal flatulence, may be noticed. When the pain is more severe the expression becomes drawn and anxious, the complexion pale, with beads of cold perspiration appearing on the forehead, and extremities cold. The pulse usually becomes small and infrequent, and in cases of lead intoxication very hard. Sometimes, however, no change is to be noticed, and this may be significant of the hysterical nature of the attack. The abdomen is retracted or distended either generally or locally, according to the cause, but during the paroxysm it is remarkably hard from contraction of the muscles, and the testes may be retracted from over-action of the cremasters. When the pain is at its worst the patient may pass into a condition of collapse, or may faint, or convulsions may be set up in children. Painful contractions of the rectum and bladder, causing urgent micturition, may be complained of. Vomiting, eructations, and the passage of flatus may lead to relief, and in the intervals between the attacks nothing beyond a feeling of exhaustion may be experienced. Where enteralgia is an entirely neurotic manifestation other nervous symptoms will most probably coexist.

Whilst in very many cases pain in the belly is but one of the symptoms a patient presents, it not infrequently happens that it is the only one, or at least the most prominent; recognition of the cause then becomes difficult, and the diagnosis only probable. Examination of the abdomen and of the motions for evidence of structural disease; the situation and direction of the pain, its character and intensity, and whether or not relieved by pressure;

the history of the attack or of any previous similar ones; the ingesta, state of the bowels, and vomiting; the temperature; the presence or absence of other nervous symptoms; and the age, sex, and condition of the patient have all to be considered, the first object being to ascertain the existence or otherwise of a definite lesion.

The term "appendicular colic" has been used to describe paroxysmal attacks of griping pain originating in the appendix in those cases where no naked-eye lesions of this structure have been observed. Careful microscopical examination, however, shows ulceration of the mucosa and bacillary invasion of the wall. The term, therefore, though it may be convenient clinically, should not be allowed to suggest the existence of muscular spasm only; but the existence of real disease, which at any moment may become most serious, must be realised.

A peculiar form of gastric pain is known as *heart-burn*, *acidity*, or *cardialgia*, caused by the contact of the acids of the stomach (whether the hydrochloric, or the organic acids, especially butyric, produced during digestion) with the surface of the œsophagus, which is normally much more sensitive than the stomach. The condition which permits this contact is a relaxation of the cardiac orifice with slight eructation of the stomach contents. It is usually associated with gastric flatulence, which favours this regurgitation, and is relieved by free eructation, or by assuming the erect position. It is not to be regarded as necessarily indicative of hyperchlorhydria nor of increased acid fermentation, and often is not so.

Closely connected with unhealthy states of the stomach and its contents are a *foul breath* and *nasty taste in the mouth*, of which there are many varieties. Ill-smelling gases liberated in the process of digestion, some of which have escaped upwards from the intestines through the pylorus, account for the first, and in some measure for the second, which is also partly due to abnormal products of digestion or of metabolism which are excreted in the buccal fluids. The condition of the mouth itself, carious teeth, etc., must not be overlooked in this connection.

Another subjective sensation is *nausea*, which is especially likely to be called forth by the sight or thought of food, or may be felt apart from such association, and is always combined with an impaired appetite, and readily passes on to vomiting. It is usually due to some altered state of the mucous membrane of the stomach, and is hence a frequent though not invariable symptom of catarrh,

both acute and chronic, but its precise pathology is not certain. It is also induced in a reflex manner by morbid conditions of the eyes, pharynx, or nose, or by pregnancy. Hysteria and neurasthenia, as well as certain toxæmias, may give rise to the feeling.

(ii.) The chief *OBJECTIVE MANIFESTATIONS* of disease of the stomach and intestines, besides such as are ascertained by physical examination, are flatulence, eructations, vomiting, diarrhœa, and constipation.

Flatulence, meteorism, tympanites.—The nature and origin of the gases normally found in the gastro-intestinal tract have been described on p. 27. In health the formation and removal of the gases—the latter chiefly by absorption and subsequent excretion by the lungs—so balance one another as to prevent undue accumulation. But from some source—the swallowed air, the carbohydrate fermentation, or transudation from the blood—the quantity may be considerably augmented, and when it is so far increased as to cause distension of the abdomen, or is eructated or passed per anum in appreciable amount, then flatulence is understood to occur. When the enlargement of the belly from wind attains enormous proportions, the gas being then sometimes free in the peritoneal cavity, meteorism or tympanites is said to exist. Defective removal—absorption—will contribute to the same result, but it is scarcely so important as excessive production.

Such conditions as bring about an undue frequency of the act of deglutition between meals will favour the intake of a greater quantity of air than is usual. Thus heart-burn insensibly leads for its relief to a constant swallowing of saliva in which much air is mixed; violent sobbing will determine an excessive gulping down of air; but cases of sudden and often extreme distension of the stomach from this cause are met with in hysteria, after attacks of migraine and other severe pain, and allied states. Such patients acquire the habit of swallowing great quantities of air, or, as Dr. J. Wyllie described in the Edinburgh Hospital Reports for 1895, of creating a negative pressure in the œsophagus by muscular effort, and thus sucking air into the stomach. A similar power of aspiration by the anus is possessed by some individuals, even to a very large extent. The ingestion of aërated and effervescing fluids is not so potent a cause of flatulence as is often supposed. The amount of gas in the stomach may be abnormally increased by the passage of gases from the intestines upwards through a relaxed pylorus. Thus it is that sulphuretted hydrogen and marsh gas, which are not produced in that viscus, may be eructated. The

latter, as well as hydrogen, which may also travel upwards from the bowels, are inflammable, and patients have been alarmed by seeing their "breath catch fire" when lighting their pipe. Eructations of these gases have frequently been noticed to be associated with or alternate with the regurgitation of acid fluids (pyrosis).

Since the fermentation of carbohydrates, whether in the stomach or bowels, is the chief cause of flatulence, it follows that this may be due to an excessive ingestion of these materials, or, as regards the stomach, to such an error in their digestion as permits a longer delay of the food in that organ than should be, or a deficiency of hydrochloric acid, by which these fermentations are held in check. In the intestines, the decomposition of the fats contributes to the formation of CO_2 and H, and owing to improper fermentations this may exceed the normal. Certain articles of diet especially favour the formation of different gases, as for instance the large amount of CH_4 derived from peas, but curious idiosyncrasies are to be observed even among healthy persons in regard to the degree of flatulence developed from a similar dietary. Foods also which contain much carbonates will liberate CO_2 on meeting the acids in the canal; and the alkaline intestinal fluids if regurgitated into the stomach will behave in the same way.

Excessive transudation of gases from the blood, or deficient absorption of the gases from the bowels, are responsible for those conditions of meteorism associated with impaired circulation in the walls of the gut, such as are seen in cardiac and portal obstruction, or in an extreme degree in blocking of the mesenteric vein. If there coexist such conditions as favour the production of gas from the contents of the canal, the resulting tympanites may be enormous, even to causing rupture of the intestine or abdominal wall. Conceivably such diseases as lead to destruction of the columnar epithelium will interfere with the proper absorption of gases, as of the digested food.

Various circumstances increase the effect of these several causes of flatulence, such as constipation or more pronounced forms of obstruction, paralytic distension of the bowel from nervous states, hysteria and the like, acute peritonitis, or œdema of the gastro-intestinal musculature. Conversely an increased peristalsis is less likely to allow of the accumulation of gases.

From the foregoing account it will be seen that flatulence may be very acute and transient, or paroxysmal, or more or less chronic in duration. Certain characters distinguish in some measure the gastric from the intestinal form, though both may occur together;

or the condition may be limited to certain sections of the bowel; but, "speaking in very general terms," as Treves observes, "it may be said that meteorism in connection with obstruction in the lesser bowel, as compared with that in the colon, appears comparatively late and is seldom of high grade." And, further, that the importance of this sign as a means of diagnosis of the seat of the mischief has been much exaggerated, although the rapid and extreme degree of meteorism developed with acute volvulus of the sigmoid flexure renders it of great value in the recognition of that particular lesion.

Ordinarily the passage of gas from one part of the tract to another, and also in some measure from the rectum to the outside, is noiselessly effected by transfusion, but in states of partial obstruction, of distension, or of irregular peristalsis, the movement is productive—and this much more in the small intestine than in the colon—of considerable rumbling sound—borborygmi—which may be accompanied by colicky pains. A belching noise, sometimes amounting almost to a roar, is caused by the compression and sudden expulsion of successive volumes of gas by the muscles of the fauces, similar to a noisy swallowing, only in the reverse direction.

The most important of the effects of meteorism are due to the impediment offered to the action of the diaphragm, with consequent effect on the lungs, and displacement of the heart; thence arise dyspnœa and feelings of suffocation, palpitation, cardiac distress, and even pseudo-anginous attacks, with engorgement of the face and suffusion of the eyes, and such remote manifestations as vertigo, dimness of vision, headache, and a special form of wakefulness, which may occur without any other sense of discomfort. As the flatulence subsides these symptoms are relieved and disappear.

Eructations.—Besides the belching up of gases, which may be sometimes very distressing in amount, coming on an hour or two after meals, or much later, some of the liquid contents of the stomach may be brought up into the mouth. These may be neutral or alkaline (water-brash), consisting generally of saliva that has been swallowed, or of the fluid remains of digestion in which the acids have been neutralised or reabsorbed. Or, as is more common, the ejecta are acid (pyrosis), whether hydrochloric or organic, and frequently associated with hyperchlorhydria, and as a consequence give rise to cardialgia. Such fluid may rise into the mouth at any time after a meal, but more often about one to three hours after. When not due to hypersecretion, the eructations usually signify some delay in the digestive process, and in the propulsion of the chyme into the duodenum.

A not infrequent phenomenon is the *regurgitation* from the stomach of small portions of partially digested food, which are either immediately swallowed again or are spat out. The circumstances leading to this symptom are various, but a relaxation of the cardia appears to be a necessary condition. It may take place quite suddenly after a fuller meal than usual, especially if there be any exertion made, but generally follows on an acute catarrh of the stomach. As a rule it is involuntary, but it may be acquired as a habit by persons of nervous temperament, and it may be so frequent as to interfere with the nutrition of the individual.

Rumination or *merycismus* differs from regurgitation in that the portions of food brought back into the mouth are remasticated before being swallowed, and there is no associated nausea. Though by no means of common occurrence, a good number of cases have been recorded, most of them being young adult males, and not infrequently is found in several members of the same family. Children have been known to acquire the habit by imitation, and it is nearly always a motor neurosis of cerebral (psychic) origin associated with a relaxed cardia. The subjects of this perverted action describe it as by no means disagreeable, some even regarding it as pleasurable, and so much under voluntary control is it in some patients that they affirm their ability to bring up at will such articles of food contained in their previous meal as they choose. Sometimes the condition develops from continued gastric indigestion which has been marked by eructations and regurgitation, the habit of reversed peristalsis becoming established. Max Einhorn, who has dealt at length with the subject (*Diseases of the Stomach*, 1898), says the duration is very variable, sometimes continuing uninterruptedly during the whole of life, sometimes occurring for recurring periods with intervals of freedom. It has been known to cease or to commence suddenly on some important change in life, such as marriage. No definite connection has been established between rumination and the state of gastric digestion; the acidity may be increased, diminished, or normal, and the motility of the stomach may be unimpaired.

Vomiting.—The forcible emptying of the stomach contents through the gullet and mouth is effected by a sudden and violent contraction of the diaphragm and abdominal muscles simultaneously, whereby the stomach is compressed, at the same time that the cardiac orifice is relaxed by contraction of the longitudinal muscular layer of the organ, the pylorus being closed. Should the cardia not be relaxed, then the spasmodic action of the trunk muscles causes retching, which is an ineffectual effort at evacuating the viscus.

Although the stomach plays no active part in expelling its contents, it is necessary that the viscus should be at least moderately distended, and hence the difficulty of vomiting "on an empty stomach." The preliminary swallowings doubtless convey a considerable quantity of air into the organ with this object. In infants vomiting is easy, the stomach, owing to its more vertical position and tubular shape, probably taking a more effective share in the performance of the act. The sickness is frequently preceded by nausea, by retching and straining, with repeated movements of swallowing, by a sense of depression, sinking, and gastric distress, together with salivation, cold sweats, and a flow of tears, but these sensory and secretory phenomena are not of invariable occurrence, and vary much in their degree when present. The pulse at the commencement of the attack is sometimes full and slow, the result of pneumogastric irritation, but often it is small, very feeble, becoming quicker as the vomiting passes off. Owing to the state of the circulation the countenance is pale, the extremities are cold, and the patient shivers. The nerve centre whence emanate the impressions that excite these movements is situated in the floor of the fourth ventricle, in close association with the respiratory centre. The efferent impulses travel by the phrenic, intercostal, and lumbar nerves, and the vagi. Excitation of the centre may follow from certain brain states and diseases which may be called central; or the centre may be reflexly stimulated from many parts of the body, and this affords a convenient basis on which to classify the various causes of vomiting, as follows:—

A. Central

Mental and emotional (psychical) states.
Hysteria, neurasthenia (so-called "nervous vomiting").
Migraine.
Cerebral growths. Meningitis. Apoplexy.
Menière's disease. Sea-sickness.
Diseases of the spinal cord. Tabes (gastric crises).

Toxæmia. Onset of many acute infections, especially in children.
Alcohol. Drugs—emetics, such as Apomorphine. Emetin.
Uræmia.

B. Reflex

Severe pain. Eye strain. Disgusting smells, sights, or tastes.
Irritation of fauces or back of tongue, or of œsophagus. Enlarged uvula.

Gastric states. Irritant ingesta. Effused blood, or tepid water in bulk.
Catarrh, acute and chronic. Ulcer. New growths.
Gastroectasis. Peritoneal gastric adhesions.
Gastrodynia.
Diseases of the intestines, *e.g.* acute obstruction.
Intestinal worms.
„ „ pancreas—acute pancreatitis.
„ „ liver and gall-bladder—passage of gall-stones.
„ „ kidney—renal calculus.
„ „ uterus and ovaries. Pregnancy.
„ „ bladder—cystitis.
„ „ testis—injury.
„ „ peritoneum.
Proptosis of abdominal viscera. Floating kidney.

Excessive coughing. Whooping cough. Advanced phthisis.
In early phthisis from presence of enlarged bronchial glands.

Addison's disease.

It is not claimed that the foregoing is an absolutely exhaustive list of the conditions with which vomiting is connected, nor that a sharp line between central and reflex causation can in all cases be drawn; thus, for instance, the morning sickness of chronic alcoholism, though mainly due to the gastric catarrh which it gives rise to, is also in part probably of a toxic character, and so central in causation, like the associated tremor and inco-ordination. But it serves to show the wide range of states by which the act may be induced, a very large number being remote from the stomach.

In respect to the diagnostic significance of vomiting, the following points should be considered—(*a*) The time relation of its occurrence to the taking of food, that is, how soon after food, whether directly or after an hour or two, or after many hours; or whether quite independent of food, and occurring on an empty stomach, or whether periodic. (*b*) The frequency, whether it occur but once or twice, or whether repeated again and again at longer or shorter intervals. (*c*) The immediately associated or preceding symptoms, such as nausea, salivation, retching, and pain. Any or all of these may be present in varying degrees of severity, or altogether absent. (*d*) Of considerable importance is the effect on the patient of the sickness, especially if this be prolonged. Should the ingesta be of an irritant character, their ejection will be likely to be followed by the relief of the pain to which they give rise. The effects of con-

tinued vomiting and retching on the circulation should be observed, and whether the pulse fail in volume, tension, or regularity. Whether also there are signs of prostration and collapse, cold extremities, pallor, sweats, and a falling temperature. The most diverse manifestations of these phenomena are to be met with in vomiting from different causes. If the sickness be repeated over long periods, for months even, the general nutrition may suffer, or there may be no bodily wasting. (*e*) Lastly, the characters of the vomit must be inquired into—the quantity, the appearance, the condition of the food, and the extent to which it is digested; the odour, the reaction, the presence of bile, blood, whether bright or altered (coffee-ground), pus, fæcal matter, parasites, or other foreign bodies.

Although it is not to be supposed that the vomiting due to each of the conditions enumerated is marked by such characteristics as to enable its cause to be at once recognised, there are nevertheless certain features which in a measure serve to suggest its origin. Thus, speaking generally, when vomiting results from some gastric condition, it bears a definite relation to the ingestion of food, although the morning sickness of the alcoholic occurs on an empty stomach, except for the swallowed saliva. When associated with pain at the epigastrium, and especially if this be relieved by the emptying of the stomach, a local lesion is to be suspected. But the same statement may be made in respect to migraine which is a cerebral affection. As a rule, also, the vomiting of gastric origin is more difficult, and is accompanied by much retching. Reflex vomiting, on the contrary, is generally easier, pain is much less often complained of, and it is more or less independent of food. When of sudden occurrence, with few other symptoms beyond a rise in temperature, the onset of an acute infection is to be feared. Cerebral vomiting is usually easy, and causes but little distress, though in the gastric crises of tabes the most violent and copious sickness and retching take place. In gastric ulcer the vomiting generally occurs very shortly after the food is swallowed, though it may be delayed, and this especially in malignant ulceration in the neighbourhood of the pylorus; in gastroectasis, the emptying of the stomach commonly takes place once in twenty-four hours, when enormous quantities of fermenting material are brought up easily but very forcibly. Vomiting of an hysterical nature offers much variety in its characters; generally independent of food, or most capricious in respect to certain articles of diet, it may be immediately excited by a meal. Oftenest easy and painless, it may be the reverse; and, occurring at most irregular times, it may on the contrary be definitely periodic.

It is seldom associated with any special disturbance of the circulation, though it has been known to cause great prostration, and repeated as it may be daily for weeks, months, or even years, it may lead to little or no wasting, and this last circumstance is eminently characteristic. Further reference to this subject is made in the description of the several diseases.

Special mention must be made of a form of periodical vomiting which, not of common occurrence, presents in the regularity of the attacks a very distinctive character. Apparently sometimes a primary neurosis, at others it would seem to be of reflex origin. The attacks occur at definite intervals of several weeks, the patient being in good health, and are violent both in their severity and the quantity of the ejecta. They are accompanied by nausea, marked gastralgia, and often pains in the limbs, and are frequently associated with headache, perhaps slight delirium, and a dry coated tongue and thready pulse. The resemblance to the gastric crises of tabes, or to migraine, *i.e.* to vomiting from some central nervous rather than a gastric cause, is obvious. In children whose brains are overtaxed at school, vomiting, with other symptoms of dyspepsia, such as stomach-ache, constipation with pallor, and a slow pulse, is not infrequent.

Sea-sickness.—This special form of vomiting appears to depend upon sudden and irregular disturbances of the equilibrium, particularly such as determine a sinking of the body. Hence it is that the pitching of the ship is more provocative of the condition than mere rolling, whilst a combination of the movements is still more effective. For the same reason swinging, and even a descent in a lift, may lead to the same result. No doubt many circumstances connected with a moving ship act as contributory causes, such as the sight of the moving water, the swaying objects on board, the stuffy atmosphere of the cabins, the smells, but these are all subsidiary to the essential one already stated. Certain personal conditions predispose to sea-sickness, and influence its severity. Thus children and old people suffer, as a rule, but little or not at all; women are said to be more liable than men, and they are likely to be more prostrated and longer in recovery. A potent determining circumstance is apprehension, which may be sufficient in itself to induce the preliminary retching. The state of the digestive organs, previous constipation, too much or too little food, or an undue liability to vomit, may contribute to the same end. Most of these subordinate causes are in abeyance during sleep, when the sickness may commence, should, for instance, the movements of the ship

have become increased during that time; this further confirms the view that disturbance of the equilibrium of the body is the real cause. Much difference of opinion exists as to whether this disturbance acts primarily on the cerebral centres, or that these are excited or inhibited from the abdomen. In connection with this question may be mentioned the occurrence of vertigo and vomiting in labyrinthine disease, and, on the other hand, the association of that awful feeling of sinking, which constitutes one of the most dreaded features of sea-sickness, with proptosis of the stomach and other abdominal viscera, and that in some measure takes a share in that symptom-complex known as "abdominal distress." In considering the pathology of this malady, its singular capriciousness must be remembered; thus some persons never suffer even to the slightest degree in the heaviest weather, others will succumb in the calmest, and it is exceptional for any not to recover in the course of four or five days, though cases of persistence for weeks are met with, and for it to be fatal is not unknown.

For an account of the phenomena of sea-sickness the writer cannot improve, either in brevity or completeness, on the following, given by Dr. Montague Murray in the third edition of Quain's *Dictionary of Medicine*:—"A slight sensation of chilliness accompanied by pallor is usually the earliest symptom. Almost simultaneously with this the sufferer is conscious of feeling ill at ease, and of a desire to remain absolutely undisturbed. Unpleasant sensations, as of a tight band over the forehead, and of retraction at the epigastrium, especially marked in each descent, are soon experienced. The breathing becomes irregular, owing largely to an unconscious fixing of the muscles at the time of any sudden change of movement. Regular, deep breathing, if persisted in, may postpone the progress of the symptoms for a short time; but sooner or later the feeling of chilliness becomes rapidly worse, giddiness supervenes, and an attack of vomiting and retching soon follows, to be succeeded by a sensation of returning warmth, accompanied by dilatation of the cutaneous vessels, and slight uniform improvement. The former condition, however, soon recurs, the subsequent attacks of vomiting being progressively worse, and the intervals between them shorter."

The vomiting first empties the stomach of such material as it contains—food and mucus—and afterwards of frothy saliva, which is bile-stained, becoming more and more bilious in character. Except in children, when the vomiting is easy, sudden, and causes but little disturbance, the sickness and retching are accompanied with a

feeling of great distress, which deepens until one "becomes indifferent alike to circumstances and fate, and feels inclined to wish that death itself might end his sufferings" (Dr. J. R. Stocker in *Allbutt's System*). A condition of profound exhaustion may be induced by the prolonged vomiting, and even fatal collapse is not unknown. In voyages of only a few hours' duration a profuse diarrhœa is not infrequent, but after a time constipation becomes more marked, as it frequently is in the sea air, whether ashore or afloat. The urine is apt to be scanty, and causes smarting in its passage. In some people the sickness may be very moderate, or even quite absent, the headache, vertigo, and sensation of depression and despondency with epigastric sinking being the chief symptoms, with great cardiac weakness, amounting even to syncope, though this is not necessarily more the case in the subjects of heart disease. Death may occur from this cause.

As a rule, recovery takes place soon after landing, or, in the case of long voyages, after a few days. Some giddiness may be experienced for days afterwards. In very severe cases the exhaustion and prostration, even to a semi-comatose state, may last for days or throughout the voyage. Women may develop hysterical attacks, or if pregnant abort, or if suckling the secretion of milk may cease; in others, menorrhagia may be set up. As an occasional result a febrile reaction may follow the attack, and sometimes it may be weeks before the patient fully recovers from the effects.

Diarrhœa.—By this term is denoted an abnormal frequency of defæcation, the motions being of a loose or liquid character. It directly depends on (*a*) an increased intestinal peristalsis, and (*b*) an undue fluidity of the bowel contents, both factors being usually combined.

The circumstances leading up to these fundamental conditions may be thus grouped:—

(*a*) *An increased peristalsis*, due to excitation of the musculature either, i., directly by the intestinal contents; ii., by lesions of the intestinal walls; iii., by irritants which reach the neuro-muscular apparatus by the blood supply; or, iv., by a perverted innervation centrally or reflexly induced.

i. The ingesta are obviously the chief source of those materials which by their irritating properties determine an increase of the intestinal motility. Such, for example, are mechanical irritants like fruit seeds, vegetable fibres, husks, and the like; poisonous substances such as arsenic, antimony, and the cathartic drugs generally; toxins of bacterial origin or derived from the putrefaction of proteid

aliment—ptomaines, albumoses, etc.—whether these be taken in as such with the food, or are the products of abnormal decomposition in the intestinal canal (autogenetic). The physical characters of the fæces, as, for instance, hard scybalous masses, may lead to increased peristalsis by the irritation they produce, and this is frequently the case after the relief of the fæcal impaction. A very excessive flow of bile may excite an over-activity of the bowels and diarrhœa.

ii. Lesions of the intestines, such as inflammation, ulcers, and new growths, apart from the irritation to which they may be supposed directly to give rise, probably increase the normal susceptibility of the canal to the reception of stimulant impressions derived from the contained material, however bland that may be. It may, however, well be conceived that these morbid states, by interfering with the due digestion and absorption of the contents, would give rise to more irritating substances than are normally produced, and hence the simplest ingesta may become harmful.

iii. Since it is known that certain materials injected into the blood-current will set up a diarrhœa, and also that the same symptom may complicate some acute infective diseases in which there is no intestinal lesion, it remains doubtful how far many of those agents enumerated in the first group may not act after absorption, rather than directly on the intestine. Or again, it may be that some toxic substances subsequent to their entrance into the blood may be secreted therefrom by the intestinal mucosa and then cause diarrhœa by the irritation they produce.

iv. The nervous control over the intestinal peristalsis may be disturbed by causes which are of central origin, of which emotional states are the most frequent. It is possible also that some poisons act in this way rather than on the periphery. Such conditions as fatigue, extreme heat, and chill, which are frequently followed by diarrhœa, may produce that effect by inhibiting the vasomotor centres and thus modifying the vascularity of the intestine and so affecting its peristalsis. Their mode of action, however, is very uncertain.

For an excessive peristalsis to be productive of diarrhœa, whatever its cause, it is essential that the colon should be implicated as well as the small intestine; although there may be reason to suppose that in the condition known as "peristaltic unrest" (p. 226) the small bowel may alone exhibit increased activity, there will not then be any diarrhœa.[1]

[1] In view of the acknowledged difficulty of explaining the propulsion of the contents of the large bowel by peristalsis only, Dr. Leftwich, in a paper published in *The*

(*b*) *An excessive fluidity of the bowel contents* follows from a diminished absorption of water—which mainly takes place in the colon—or from an excessive intestinal secretion, the two conditions frequently coexisting.

The mere hurrying on of the material by an increased peristalsis interferes with the passage of the fluid into the vessels, and this would be further aided should the mucosa be atrophied or extensively ulcerated, the absorbent surface being thereby damaged.

Very often there is a hypersecretion from the mucous surface, such as may be induced by saline or other hydragogue cathartics; sometimes also a passive congestion of the intestine from hepatic cirrhosis or cardiac disease may be relieved by a discharge of fluid into the bowel, although this condition is oftener associated with constipation, from the hindrance it offers to the activity of the muscular coat. Or the hypersecretion may be a neurosis, coincident with a peristaltic over-activity due to a like cause. The action of some toxins—as, for example, that of cholera—may be explained by their effect on the neuro-secretory mechanism.

Critical diarrhœas, which may accompany the crisis of an acute illness as pneumonia, are probably associated with excessive secretion into the canal. The occurrence of a diarrhœa is favoured, even if not entirely caused, by such conditions as may interfere with the integrity of the sphincter ani, such as a ruptured perineum or a growth just within the anus.

Although set forth here in categorical fashion, it must be remembered that many cases of diarrhœa owe their origin to a complex causation, and this is especially so in infants and children in whom the age is a powerful predisposing circumstance. The connection of hot weather with diarrhœa is also a complicated one, in which the ingesta, the digestive power, the vascularity of the canal, and the greater probability of the presence of micro-organisms are all concerned.

From a review of these causes it is clear that a diarrhœa may in its course be acute or chronic, may be associated or not with pain,

Edinburgh Medical Journal, Dec. 1902, suggests that this portion of the canal should be regarded as a syphon, the short limb of which is formed by the ascending colon, and the long limb by the descending and sigmoid colon and rectum. That the bowel can so act he has demonstrated experimentally even when the contents are partly fluid and partly gaseous. That some such syphonage may take place when the colon is full of fluid would seem possible, and the violent, sudden, and complete discharges that occur when the bowel is so filled are quite consistent with such a view. "In diarrhœa the time occupied by the refilling may be very short, and the syphon action recur at frequent intervals."

may be the predominant symptom or but one of many, may have a direct relation to the taking of food, and it may be up to a point beneficial by removing noxious material. The evacuations may be but few in number and the attack pass off with complete recovery, or they may be almost incessant, as in cholera and dysentery, lasting for several days, or several loose motions may continue daily for weeks or months. A limitation of the discharge to the morning, comparable to the vomiting, is characteristic of chronic alcoholism, and the same has been noticed in malignant and syphilitic diseases of the rectum, much discomfort being experienced in the earlier part of the day with subsequent freedom.

If long continued, as in chronic colitis, or if very excessive over a short time, as in cholera, the nutrition suffers considerably from the interference with digestion and absorption that the exalted motility of the bowels produces; but the degree of deterioration of the general health which a diarrhœa determines is largely conditioned by the nature of the underlying cause, as well as the presence of other symptoms, such as pain. A very fair degree of well-being may coexist with a long-continued looseness of the bowels.

The value of this symptom as an element in diagnosis is chiefly based upon the character of the stools, which vary from a "rice-watery" fluid to a pultaceous consistency, may be colourless, ochre yellow, all shades of brown, or green, may contain large quantities of mucus, or apparently none, blood in variable amount and appearance, milk curds and sometimes recognisable portions of food that have entirely escaped digestion (lientery).

Constipation.—This—"one of the commonest troubles of civilised life"—is best defined as "an insufficient emptying of the bowels." It will be noticed that in this definition there is no specific reference to frequency, still less does it imply a daily action of the bowels; it states nothing as to time, but only as to completeness of action. It is important that this should be realised, for there may be a daily action and yet there may be constipation, because that action is inadequate. There is a popular notion that in the healthy individual the bowels must of necessity move daily, and in most healthy people it is doubtless usual for this to be the case, but it is not absolutely necessary, and in treating constipation it is not by any means requisite that this daily action should be aimed at if it have not previously been the habit of the individual. It must be remembered that habit enters very considerably into this function, and the daily evacuation which is the case with most people is largely the result of habit rather than of necessity. There is nothing in the

construction of the bowels that makes it imperative that they should be emptied every day, and whilst many individuals suffer much or little if they are not, there are others in whom this evacuation may not take place oftener than once in two, three, or four days, and who yet are quite healthy, and may even be not so well should there be an action every day. The essential feature, then, is adequacy and not frequency. When asked if their bowels are open every day, many persons will say, "Yes," and amongst a certain class, however true this may be in fact, it may still be consistent with a state of constipation; the bowels have been opened, but the action is not adequate, and there is a constantly recurring residuum. This is frequent in all women, among whom it is a characteristic to be constipated.

In seeking to obtain a comprehensive view of the causes of constipation, account must be taken of the nature of the act of defæcation. This act is the resultant of three separate muscular efforts, viz. the peristalsis of the intestines, the contraction of the abdominal muscles, the diaphragm being fixed in inspiration, and the relaxation and drawing up of the sphincter ani. Each phase has its separate nervous control (p. 30). The normal stimulus to the act is found in the character and bulk of the intestinal contents—solid, liquid, and gaseous. An ordinary fæcal evacuation weighs normally about five to six ounces, and contains about 75 per cent of water, and if this proportion to 25 of solid matter be altered, in one direction diarrhœa, and the other constipation, may result. The bulk of the intestinal contents, or rather the degree of distension of the canal, is largely influenced by the flatus present, of which a certain amount is the normal condition, and favours the action of the bowels. As regards the solid contents, certain elements of the food contribute to call forth the action of the bowels, such as the indigestible fibres and husks of certain kinds of grains and fruits and vegetables, which are in some measure irritants. In this connection also it must be remembered that some of the salts of vegetables and fruits are laxative in their action, and these play their part in the stimulation of the bowel. Possibly also the changes that take place in the intestinal contents due to the action of bacteria are of an irritating nature, and excite a peristaltic action. Formerly much importance was attached to the action of the normal secretions along the course of the intestinal canal, especially the bile. Probably when this latter is prevented from getting into the intestines there is a liability to constipation; the exact part which it plays is not known, though it seems to be a very subordinate one, since very

definite constipation may coexist with a free flow of bile. How far the question of habit—that is, an action the efficient performance of which is acquired by constant repetition—may have determined in the nerve centres a rhythmic action cannot be affirmed, but it may probably have some influence.

The several phases of the act of defæcation may be temporarily inhibited by conditions such as anxiety and other mental and emotional states or distinct diseases of the nerve centres.

A convenient, though quite artificial and arbitrary, grouping of the various causes of constipation, or the conditions with which it is associated, is the following, modified from that suggested by Sir Lauder Brunton :—

1. Those due to a diminution in the expulsive force.
2. Those due to increased resistance to the normal expulsive force.

And it not infrequently happens that the explanation is to be found in both these groups.

1. (*a*) *Affections of the peristaltic mechanism, or the intestinal neuro-muscular apparatus.*—Degeneration or other states of malnutrition of the muscular coat lead to impaired peristalsis. Thus constipation occurs in peritonitis, and sometimes in enteritis, from the inflammatory process involving the muscular layers, and is frequent in anæmia and states of general enfeeblement. Engorgement and œdema of the intestinal wall, as in hepatic cirrhosis or obstructive heart disease, will produce the same result.

It is also found that some cerebro-spinal states are apt to be complicated with constipation, both those in which the lesion is gross, such as meningitis, apoplexy, and tabes, and others in which no structural defect is recognisable, as hysteria, neurasthenia, and states of anxiety or overwork. Possibly also the confined bowels may be due to causes acting reflexly on the intestinal musculature.

Poisons, of which lead is the commonest, may cause constipation by morbid changes induced by them in the intestinal nerves. Other drugs, such as opium, and still more frequently the continued use of aperients, whether cathartics or salines, which, though acting differently, tend to the same result, bring about a progressively diminishing peristalsis, and are among the commonest causes of chronic or habitual constipation. Repeated distension of the lower bowel by large enemata has the same effect.

Atony of the intestine—a convenient term expressive of neuro-muscular enfeeblement—leads to dilatation, and a number of cases

of constipation owe their origin to this condition. The opposite condition, namely spasm, is sometimes the cause.

(*b*) *Weakness of the abdominal muscles*, whilst rarely solely responsible, is often a potent accessory cause of constipation. Large accumulations of fluid or tumours in the abdomen interfere with the efficient contraction of these muscles on the intestines, and so impair the expulsive force. In great laxity of the abdominal walls from repeated pregnancies or tappings and in extreme obesity the muscles contract at great disadvantage, and most inefficiently.

Inasmuch as the daily evacuation of the bowels is so largely a matter of habit, any interference with the regularity of that habit is liable to cause disturbance, and this chiefly in the direction of suppression. Thus it is that a neglect of the desire for stool, especially if this be constant, or may be on a single occasion, as when travelling, is sufficient to set up a habit of constipation. A frequent cause of such neglect that demands careful inquiry for, is the fact that an action is accompanied with great pain—from fissure, piles, or a growth in the rectum, which leads to deferring the effort as long as possible. Among other circumstances that tend to impair the neuro-muscular irritability and contribute to induce constipation are want of exercise and sedentary habits, and in women tight lacing, whilst the normal stimulus to contraction may be lacking from insufficiency of food, or of the indigestible residues of food which furnish with epithelial debris and bacterial remains the bulk of the colon contents. Individual idiosyncrasies are met with, certain articles of diet producing constipation in some persons, as other articles may cause diarrhœa. The bowels are not infrequently confined in the course of gastric affections, such as ulcer and disturbances of the acid secretion, and particularly in cancer of the stomach. Since vomiting is commonly associated with most of these conditions, they may be presumed to act by causing a decrease in the intestinal contents and probably at the same time interfering with the proper nervous control of the musculature. It is not very unusual to find constipation the characteristic of many members of the same family, even in several generations, and this must be regarded as due to a defect in the neuro-muscular activity.

2. *An increased resistance to the normal expulsive force* is to be found in (*a*) morbid states of the bowels, or in (*b*) the character of their contents, conditions which in the present connection may be termed "mechanical."

(*a*) Such *structural changes* in the intestinal canal or displacements of certain sections of the intestines as offer obstruction to the

onward passage of the fæces (p. 247) are obviously to be reckoned as causes of constipation. Some of these are acute in character and short in duration, as, for example, a strangulated hernia, and in many of these the constipation is but one of many symptoms, and is not the main or essential affection, as it frequently is in many of the causes already referred to, or as it is in displacements of the transverse or sigmoid colon, which in adults are the only portions of the large intestine provided with a mesentery, and in atony and dilatation of the canal. Congenital constipation is often due to a long and displaced descending and sigmoid colon. Pressure on the bowel by abdominal tumours or adhesions fixing coils of the intestine to each other or to the abdominal wall are, unless the latter be very extensive, but exceptional causes of constipation, since such conditions do not paralyse any length of the muscular coat which is requisite to arrest the peristalsis.

(*b*) *Character of the intestinal contents.*—Just as these when too scanty may fail to provide the necessary stimulus to contraction of the musculature, so an enormous accumulation in the colon may arrest its action and cause paresis of the gut. It is, however, the consistency of the fæces rather than the bulk which is the chief factor in producing constipation. It is said that if the proportion of water in their composition should fall from the normal 75 per cent to 30 that the peristalsis would be completely ineffectual, and a diminution to 50 per cent would considerably interfere with their propulsion through the colon and their extrusion therefrom. Astringent drugs, by causing a decrease in the secretions of the mucosa, leading in consequence to a dryness of the fæces, are efficient in suppressing the activity of the bowel, some also acting directly on the neuro-muscular apparatus. Among very exceptional obstructions caused by the nature of the intestinal contents are concretions, large gall-stones that have ulcerated from the gall-bladder into the gut, and accumulations of fruit seeds, vegetable husks, or hairs.

It is not always easy to distinguish those **symptoms** which are due to the actual constipation from those of the condition which has caused the constipation itself. Moreover, it is very certain that the bowels may not have acted for many days, even for weeks, without giving rise to urgent symptoms; whilst, on the other hand, the missing of one day may be fraught with considerable discomfort. When the constipation is habitual a state of somewhat impaired health often becomes established which the patient tolerates unnoticed, now and then interrupted by an exacerbation of some of the symptoms.

All degrees of abdominal fulness and uneasiness are complained of, perhaps amounting to a feeling of dull weight or pain, or even attacks of colic from spasmodic contractions of the bowels. The amount of distension is very variable; there may be an enormous accumulation of fæces in the colon with only very moderate fulness of the abdomen. Should, however, as is usual, there be much flatus, then the belly is necessarily much bigger. Manipulation may allow the detection of scybala, oftenest situated in the sigmoid, descending, or transverse colon or cæcum, sometimes movable lumps of the size of a walnut or much larger, and possibly undergoing alteration in shape by pressure; sometimes, however, and particularly in the iliac fossæ, the masses are more fixed and indistinguishable by palpation from new growths, with which of course they may coexist. Very definite evidences of gastric indigestion frequently coexist with very confined bowels, and may be at once relieved, and later entirely removed, by measures solely directed to correct the constipation. The more prominent indications of this condition are loss of appetite, a furred tongue with foul breath and a nasty taste in the mouth, a dry skin and slightly icteric conjunctivæ, and especially discomfort after meals with flatulence. With these symptoms, which are directly referable to the alimentary tract, other evidences also occur. These particularly concern the nervous system, such as headache, vertigo, irritability of temper, depression, and hypochondriasis. It will, however, be more convenient to consider these remote symptoms later, together with those in which the circulatory and respiratory symptoms are concerned, as well as the question of their being due to auto-intoxication (p. 232). It may, however, be pertinent to observe that the occurrence of these distal symptoms appears to be closely related to the fluidity of the retained intestinal contents, in which putrefaction more readily takes place and the products are more easily absorbed, than when the fæces are of the nature of hard scybala.

Other neuro-motor impairments.—Besides such very obvious disturbances of gastro-intestinal motility as regurgitation, vomiting, and diarrhœa, there have been described certain other perversions of the motor functions, especially affecting the orifices of the stomach. A *spasm of the cardia* and of the *pylorus*, and also a *paralysis* of the musculature of these apertures, is spoken of, the former determining an obstruction, as the latter causes an insufficiency or incontinence. It must be admitted that the evidence for the existence of these conditions is at present far from certain, and they are for the most part clinical assumptions rather than demon-

strable facts. They are regarded as (*a*) the manifestation of neuroses such as hysteria or neurasthenia, and pyloric incompetence has been stated to have occurred in compression myelitis; or (*b*) secondary to such structural lesions of the stomach or duodenum as inflammation, ulcer, or new growths. The spasmodic affections are also attributed to hyperæsthesia of the gastric mucous membrane, to hyperacidity, and to flatulent distension of the stomach.

The **symptoms** suggestive of spasm of the orifices are those of obstruction more or less complete, either of an acute transient character or of a more chronic duration, interfering with the proper passage of the food into the stomach or out of that organ into the duodenum. Pain is not a constant phenomenon. The passing of a stomach tube may disclose a cardio-spasm, or it may be recognised by auscultation for the deglutition sounds; "absence of the second deglutition sound means an obstruction at the lower end of the œsophagus or at the cardia" (Hemmeter). The delayed appearance of iodine in the saliva after the administration of $\frac{1}{10}$ gramme of iodoform in a gelatin capsule during a meal, may be taken to indicate pyloric obstruction (spasm). The paralytic states of the apertures permit of an escape of the gastric contents with undue facility, whether onwards through the pylorus or backwards into the gullet, causing regurgitation. The imperfect gastric digestion of the food which its too rapid escape into the duodenum would lead to, might be compensated for in the small intestine and the body nutrition be maintained. But the unusual material in the bowel might lead to diarrhœa, and a sense of emptiness and hunger is apt to be frequent. Insufficiency of the pylorus may be detected by Hemmeter's method of duodenal intubation. It is asserted that spasm of cardia and pylorus may coexist, and very rarely the entire stomach may be involved, the organ feeling to the observer as a hard ball.

A condition characterised by excessive movements of the stomach, termed by Kussmaul "*peristaltic unrest*," has been described, and is certainly of more frequent occurrence than the foregoing. Setting in after a meal, or at night with an empty stomach, are rapid and forcible movements, which may be seen and felt passing from left to right should the stomach be dilated or displaced downwards. Accompanying these are sensations of discomfort in the region of the stomach, seldom amounting to pain, but rather of movements, with nausea, eructations, and even vomiting and generally constipation with flatulence. It has been attributed to undue irritability of the sensory nerves determining

reflexly the gastric movements, the stimulant being found in a hyperacidity, or in pyloric stenosis. It has also been looked upon as a primary motor neurosis. A similar condition has been described in the intestines. An excessive excitability of the neuro-muscular apparatus of the intestine may manifest itself as a diarrhœa from increased peristalsis, and the mere taking of food may be sufficient to induce an action of the bowels. The intestinal crises of tabes are severe cases of this class.

Localised spasms of the bowels accompanied by pain constitute colic, and are commonly due to indigestible material, to flatulent distension, constipation, or any condition which causes obstruction of the gut. Tenesmus, or the painful spasm of the anal sphincters, usually results from ulceration or other lesions in the rectum, and is a common symptom with an intussusception.

Deficient motor power of the stomach or intestines or of the entire gastro-intestinal canal, often spoken of as *atony*, is of frequent occurrence, and follows from various causes, among the most frequent being changes which take place in the musculature as the result of degeneration, inflammation of the stomach or bowels, chronic congestion, and œdematous infiltration. It may be a part of the general muscular weakness following on exhausting disease or continued malnutrition; and such causes as lead to obstruction of the canal will by the consequent dilatation sooner or later bring about a weakening of the muscular coat. The frequent over-stimulation of the bowels by strong aperients and the condition of constipation itself also tend to impair the contractile power. Emotional states, hysteria, and neurasthenia may be the cause in some cases, and it is said that some cerebral diseases are accompanied by symptoms of intestinal paresis, the afferent impulses passing by the vagi.

The chief results of gastro-intestinal atony are seen in the delayed passage of the contents, whether from the stomach, in which case gastric digestion is much interfered with, causing flatulence, acid fermentations, and their consequences; or in the intestines constipation being the main feature. Moreover, the weakened muscle yields more readily to the pressure of the accumulated contents than a healthy tissue, and thus favours dilatation of the organs, as this in turn leads to further atony—the one condition intensifying the other.

II. Remote symptoms.—The symptoms immediately resulting from an indigestion which are unconnected with the alimentary canal or its glands, but relate to parts and regions remote therefrom, are

both clinically and pathologically the most interesting phenomena connected with dyspepsia. Insomuch as many of them are induced by affections of other systems, they are not diagnostic of indigestion, and require careful weighing for their proper appreciation; and many here included as of dyspeptic origin are perhaps more correctly to be regarded as evidences of tissue-malnutrition to which the mal-digestion has given rise. They may be the grounds upon which the patient seeks advice, and the manner in which they may be presented is often such as not to suggest, at least in the first instance, a digestive disturbance as being at their root. Since also they may and frequently do occur without any pronounced symptoms more obviously referable to the digestive organs the difficulty of diagnosis becomes the greater, and the condition frequently comes to be regarded as one of primary nervous, cardiac, or pulmonary disease, the relation to an underlying indigestion being so obscure. Indeed the justification for their correct understanding may have to be based upon the improvement or cure which follows a treatment specifically directed to the digestive system—a not altogether satisfactory conclusion, it must be admitted. Some of the more exceptionally occurring of these manifestations appear in the writer's experience to be connected with individual idiosyncrasies in respect to certain articles of diet which to the majority of persons are quite harmless.

The remote symptoms may be thus grouped; but it is not claimed that the list is exhaustive, the more usually occurring being alone enumerated :—

Nervous.—Dulness, heaviness, throbbing of the head. Headache.
Vertigo, giddiness. Facial neuralgia.
Subjective sensations of sight, hearing, and smell.
Drowsiness, heavy sleep, torpor. Wakefulness.
Mental irritability and depression, hypochondriasis, melancholia.
Muscular twitchings. Cramps. Tetany.
Referred pains.

Circulatory.—Palpitation. Cardiac irregularity. Intermittence.
Syncope.
Cardiac pain. Angina.
Flushing of face. Coldness of the extremities.

Respiratory.—Cough. Dyspnœa. Sighing.

Cutaneous.—Various urticarial and erythematous rashes.

Urinary.—Deposits of urates, phosphates, oxalates, uric acid.
Albuminuria. Albumosuria. Glycosuria.
Indicanuria. Presence of aromatic sulphates.

The group of remote manifestations here comprehensively designated "nervous" are among the most prominent, and of these headache is perhaps the most frequent in occurrence, as in turn indigestion is probably the commonest cause of headache. As regards locality, whether generally diffused or localised to the frontal, vertical, temporal, parietal, or occipital regions, the time of its occurrence in relation to food, its character—so far as words can describe it—dull, aching, throbbing, etc., nothing specifically characteristic can be affirmed—nothing, that is to say, which can at once enable a given headache to be set down as dyspeptic in origin, however probable such may be the case. Scarcely less frequent than the foregoing, and often extremely distressing, are giddiness and vertigo. Subjective affections of sight, such as ambiyopia, coloured flashes of light, muscæ volitantes, etc., or of hearing, such as tinnitus aurium, are also of no unusual occurrence in dyspepsia. The association of drowsiness with indigestion is generally familiar, but the connection of wakefulness with the same state is not perhaps so well known. The condition of irritability of temper, contrasting so strongly as it does with the sense of comfort and placid well-being that attends a good digestion, is closely simulated in the course of those more remote disturbances of nutrition to the production of which dyspepsia undoubtedly contributes, although it is here sought to distinguish between the symptoms for which indigestion is distinctly responsible and those due to the later metabolic change in the tissues which are really different from the true digestive processes. Attacks of mental depression amounting to actual hypochondriasis and melancholia with delusions of suspicion are well recognised as of frequent dependence on imperfections in digestion; the very name melancholia, a term of Hippocratic medicine, signifies the connection. It has also been made clear that outbursts of mania or other psychic disturbances in those whose mental powers are perturbed are not infrequently traced to failures in the digestive process, especially if there be constipation. A close relationship between that condition of deficient nerve power known as neurasthenia and disorders of digestion has been described, although here again the general deterioration of nervous energy may more immediately be the expression of an impaired nutrition of which a dyspepsia may be but one factor in the production. Corresponding in some measure to neurasthenia of dyspeptic origin is a general sense of lassitude and muscular weakness of like cause, though partly due in all probability to an inadequate removal of the results of an abnormal tissue metabolism; and the same may be said for those violent

cramps, more especially of the muscles of the calf, which not infrequently result from dietetic or digestive errors.

As Dr. Head has shown, certain regions of the scalp are associated with the segmental areas of the trunk as low as the 10th dorsal; it follows that reflected pain and tenderness may be experienced over the temporal, vertical, parietal, and occipital regions, when the areas corresponding to the stomach, viz., the 7th to 10th dorsal, are involved, and as discomfort over the latter vary in intensity so will the former. The intestine from the duodenum to the sigmoid flexure is related to the 10th, 11th, and 12th dorsal segments, and reflected pain and tenderness may be present over these areas in affections of these viscera.

The mere mention of the disturbances of the circulatory function attributable to a disordered digestion is sufficient. Respiratory defects are much less commonly observed, though the so-called "stomach cough" is sometimes the expression of gastric or gastro-intestinal irritation. A true dyspeptic dyspnœa is less well known, but of its existence there is little doubt, both with other dyspeptic symptoms or so predominating as to obscure its real origin. Along with a sense of oppression and impeded breathing is a constant desire to take a deep inspiration occurring in attacks often of some hours' duration and not infrequently at night—a condition which recalls in its description an asthmatic paroxysm, as a provocative of which indigestion also takes a high place.

A connection between indigestion and affections of the skin has long been recognised, although different opinions have been held as to the degree of the relationship. For whilst it is very certain that an eruption of urticarial wheals, of erythema, of pruritus, or even of eczema or acne rosacea, to mention only those cutaneous affections of most frequent occurrence, may follow the ingestion of certain articles of diet, just as these and other rashes may be caused by various drugs, the actual conditions of their development are apt to be most uncertain. Not only are such results met with in but few persons and are of nothing like universal occurrence, but they are not constant even in the same individual, and they may or may not be accompanied by definite dyspeptic symptoms, such as pain, vomiting, diarrhœa, etc. Setting aside for the moment the question of how such eruptions are produced, it is doubtful whether they can be regarded as results of indigestion, and whether they are not rather to be considered as caused by the direct toxic action of certain ingredients contained in the articles of food with which the process of digestion is not strictly concerned. Some observers have had reason

to believe that, coincident with the cutaneous urticaria or eczema similar if not identical conditions are developed in the alimentary mucous membrane, and in such cases evidences of a perturbed digestion are of course likely to be manifested. The long-continued ingestion of certain articles of diet, such, for instance, as alcohol, may, by the injurious effect it exerts in the nutrition of the tissues, tend to produce certain skin affections, but such are not to be regarded as true dyspeptic phenomena, from which indeed they are really remote; rather are they true skin diseases predisposed to by the malnutrition which improper feeding or imperfect digestion have determined and immediately induced by some locally exciting cause. It is on the whole probable that the relationship of skin affections to dyspepsia is not so close as was formerly supposed, and that in most cases the element of idiosyncrasy has to be considered—a circumstance that would go far to shift the responsibility for any given eruption to the cutaneous structures themselves or their trophic governance rather than to place it upon a disordered digestion.

The evidences of indigestion manifested in the urine are seldom the cause of actual discomfort to the patient, and the significance of the abnormalities observed is only very vaguely understood. Even the precipitates of lithates, as well as of phosphates and oxalates, which may accompany very definite dyspeptic conditions, are probably expressions of later metabolic perversions rather than that of direct imperfections of digestion. It is very certain, however, that the process of digestion even in health does exercise a very real effect upon the character and composition of the urine, as witness the increased alkalinity after meals and the variations in the amount of the nitrogenous constituents, urea, uric acid, and creatinine, with a full or diminished diet, although such bodies as these are only remotely connected with digestion and are products of tissue waste. The kidney as an excretory organ is concerned with the elimination of some of the results of intestinal putrefaction which are absorbed into the blood in place of being voided with the bowel evacuations. The most important of these substances are the aromatic sulphates and albumoses. The former are compounds of such bodies as indol and skatol with sulphuric or glycuronic acid and alkalies, especially potash. These bodies, known as chromogens, are themselves colourless, though readily giving rise to pigments on the addition of acids. They are mainly, if not entirely, originally derived from the intestine, and their presence in excess may be regarded as an index of undue intestinal putrefaction; but their exact significance beyond this general fact is unknown. The existence of albumosuria, and the very excep-

tional occurrence of peptonuria, are indicative of a certain degree of proteid digestion, the products of which have been absorbed into the blood, and in virtue of their toxic properties have induced an irritation of the kidney and thus led to their excretion. But it by no means follows that this digestive process has taken place in the alimentary canal, for the albumins of the tissues may be so dealt with by microbic invasion. Even when their source may with reason be assumed to be the gastro-intestinal tract, their presence in the urine may not necessarily depend upon an error in digestion so much as some imperfection in the process of absorption from the mucous surface of the bowel. Glycosuria, oxaluria, and phosphaturia are probably but indirectly symptomatic of mal-digestion, whilst their relation to tissue metabolism is unquestioned. On the whole it may be said that the urinary abnormalities immediately due to dyspepsia are few, and interesting as they may be, and valuable as is their promise, they cannot at present be said to count for much in the actual diagnosis of indigestion, far less to afford any indication of the precise nature of the digestive fault.

The explanation of these exceedingly diverse and widespread results of an imperfect digestion, distinguishing them so far as possible from the effects of a vitiated metabolism, is exceedingly difficult. The mechanical interference with the action of the heart or diaphragm that flatulent distension may cause will doubtless account for many of the cardiac and respiratory symptoms; and a more precise chemical pathology will probably in time explain some of the urinary changes. Two main views are held to account for the distal phenomena of indigestion. Some, such as the flushings, are attributed to reflex irritation, whilst the greater number are set down to auto-intoxication, from the absorption of poisonous substances developed during the perverted digestion. Whether when absorbed into the circulating medium these substances produce their effect directly on the tissues or indirectly *viâ* the nervous system there is little or no evidence to show, merely surmise; but whether by either or both means such poisons would seem adequate to explain most if not all the remote symptoms which are properly referable to indigestion. That peripheral irritation of gastro-intestinal origin might of itself be sufficient to induce distal symptoms without the absorption of any harmful material appears possible, but of the suggested explanation—viz., that they are due to variations in blood pressure reflexly induced by gastro-intestinal states—very little can be said. Such symptoms as giddiness, vertigo, and even some dyspeptic headaches, may be thus explained, and some grounds for

this may be found in the circumstance that some of these phenomena may immediately disappear after action of the bowels—a circumstance that is always attended with a fall of blood pressure as constipation is accompanied by the reverse. The relief from headache may follow so quickly as almost to negative the idea that the emptying the bowel has simply produced the effect by getting rid of toxic material. Owing in great measure to the difficulty of proof, there is an indisposition on the part of many to accept auto-intoxication as the explanation of some of the more severe nervous symptoms, which are referred by others to indigestion, such as hypochondriasis, melancholia, or other forms of insanity; and some have gone so far as to attribute the associated gastro-intestinal symptoms, and even the structural changes said to be found in these organs, to the cerebral state, regarding them in this respect as secondary; and that hereditary brain weakness or instability is requisite for the toxæmia to produce any such effect.

The effect of a long-continued dyspepsia, or even of an acute attack of a severe type, will seriously impair the general nutrition of the body, interfering as it does with the due supply of proper aliment. Hence loss of flesh is a common symptom. The degree which this may reach varies with the cause and nature of the indigestion and the existence of other diseases. At the same time errors of digestion of a troublesome and obstinate character are often met with in persons who give no other obvious evidence of ill-health, and who maintain a normal weight. The lowering of the general nutrition tends to increase the vulnerability of the organism and its susceptibility to untoward influences.

The foregoing account of the many symptoms associated with defects in the performance of the digestive process shows the wide range of morbid manifestations and the many organs and functions which are liable to be disturbed. Unfortunately for diagnostic purposes it cannot be said that any of the numerous phenomena can be recognised as specially indicative of any special cause, the relation of precedent conditions to symptomatic results being so extremely complex, and so many similar symptoms being determined by totally different conditions.

Among the many features of dyspepsia that call for comment the uncertainty of its manifestations is as well known as it is remarkable. How commonly is it the case that a patient—the subject may be of frequent indigestion—consumes with impunity a meal that violates in quantity or quality most of the dietetic rules

he has been accustomed to observe and has found almost essential to his well-being. "That one man's meat may be another man's poison" all are familiar with, and even how often the food which is suitable to-day may disagree to-morrow or the plainest fare may provoke an indigestion. The fact that what are called errors in diet do not of necessity induce discomfort, local or remote, and with many people rarely, if ever, lead to indigestion, would seem to suggest that the *materies morbi* is less in the ingesta as presented than in the subsequent digestive processes, and is to a great extent independent of the actual food taken.

In the consideration of indigestion it is essential to remember the sequential and, to a certain extent, the inter-dependence of the successive stages of the digestive function; how that the gastric processes are influenced in some measure by what has previously taken place in the mouth, as they in turn modify the changes in the intestine. Hence it is that many errors of stomach or bowel digestion may be traceable to imperfect mastication or other buccal functions, and that such peristaltic disturbances as diarrhœa or constipation, although directly dependent upon the intestines, may really owe their causation to gastric inefficiency.

Groups of these various symptoms, both local and remote, are apt to occur in association with tolerable uniformity and to afford a basis of classification of the numerous forms of dyspepsia, which have been characterised by such terms as "acid," "irritative," "atonic," "flatulent," "bilious," and the like, the duration and general character of the attack justifying the further expressions, "acute," "subacute," or "chronic." It cannot, however, be maintained that such a grouping presents any scientific precision, though convenience may sometimes warrant the employment of such an arrangement. Far less open to objection is a classification based on the site of the digestive perversion, whether oral, gastric, duodenal, or intestinal, although the data for such are still very deficient. From another aspect the forms of dyspepsia may be arranged according to the particular element of the food which is ill-digested —proteid, fat, or carbohydrate. But probably the most satisfactory basis is that of causation, which would give the following forms: (i.) Dietetic; (ii.) Symptomatic, *i.e.* the manifestation of primary affections of the digestive organs — structural, nervous, and vascular; (iii.) Bacterial indigestion; and those rarer conditions due, it may be presumed, to (iv.) Defective absorption of the digesta, and (v.) To improper excretory processes taking place in the gastro-intestinal canal. At present, however, the

grounds are not sufficiently precise to admit of the general adoption of such a plan.

In any arrangement, however, of the signs and symptoms of indigestion into such groups as would permit of their being considered as special forms of dyspepsia, it must not be forgotten that the manifestations are all referable to perversions of one or more of the three fundamental phases of gastro-intestinal activity, viz. the sensory, motor, and secretory functions of the canal; and, further, that inasmuch as these very frequently depend on a morbid condition of innervation of the structures concerned, some justification may be found for regarding a large proportion of indigestions as "nervous" in character. When, however, the contractile or secreting structures have undergone definite change as the result of atrophy, inflammation, degeneration, or the like, the "nervous" element in the production of the resulting symptoms is wanting or of secondary importance. Included within the term nervous dyspepsia are those disturbances of gastric function for which no structural cause is to be found in the organ itself, but are attributed to perverted nervous influence, whether this be due to gross change in the nerve centres, such as tabes dorsalis, cerebral tumour, meningitis, etc., or to general states of these centres for which no anatomical substratum has as yet been discovered, such as hysteria and neurasthenia, or that still more indeterminate condition known as the "nervous temperament." By far the greater number of cases are referable to these last-named causes, whether they have been inherited, as is more usual, or have been acquired as the result of excesses in living, of nervous exhaustion, of chlorosis, of gout, of previous illnesses like influenza, or of other circumstances. Not a few may be traced to causes acting reflexly, especially pelvic disease, ovarian, uterine, or rectal, and even, it has been asserted, to eye strain and astigmatism.

The sensory and motor disturbances of digestion have been sufficiently treated of in the foregoing pages. There remains to be given some account of the more specially secretory errors as they are recognised in the gastro-intestinal tract, the salivary perversions having been referred to on p. 99. It is to be understood, however, that whilst either secretory, sensory, or motor defects may occur separately, they are oftener met with in combination.

The alterations in the composition and quantity of the gastric juice at present recognised chiefly concern the hydrochloric acid; of the pepsin and other ferments less is known, but they do not in all cases appear to vary *pari passu* with the acid. Thus it is found

that the quantity of gastric juice secreted may very much exceed the normal, being poured out either almost continuously (Reichmann's disease), or at intervals quite irrespective of the presence of food in the stomach. Very frequently the fluid contains a greater percentage of hydrochloric acid than in health. These conditions are for the most part met with in grave nervous states, such as the enormous secretion in the gastric crises of tabes, often lasting over several days, or the similar state observed in the rare and obscure cases of acute gastric dilatation which are usually rapidly fatal. They often appear to be quite unconnected with any obvious lesion of the stomach.

In another group of cases of more frequent occurrence the percentage of hydrochloric acid exceeds the normal (.1 to .25), and may be as much as .4 or .5, with no increase in the quantity of fluid. This *hyperchlorhydria* is a frequent accompaniment of chlorosis, and is constantly associated with gastric ulcer, with the production of which it is largely connected. Another cause for hyperacidity, quite distinct in character, is an over-formation of organic acids derived from fermentation of the carbohydrate ingesta—a true digestive fault —which is rendered possible by a deficiency in the hydrochloric acid secreted, or by a great excess of sugars and starches. It is important to recognise the difference in the nature of these two conditions, which may appear to be alike to the mere test of litmus paper.

In contrast to the foregoing are those patients in whom there is a deficiency of gastric juice, even, it is said, to complete suppression —*achylia gastrica*—or a percentage of hydrochloric acid below the normal. Although these conditions may occur without any change in the mucosa, and may be the effect of nerve influence only, they are most frequently the result of the chronic catarrhal or other alterations in the structure of the mucous membrane when the glands are destroyed, such as is particularly found in cancerous infiltrations or in cirrhosis ventriculi.

The **symptoms** which these several morbid states give rise to are necessarily closely associated with imperfections in digestion, and are in some measure modified by any coincident alterations in the motility of the stomach. All degrees of sensory disturbance are experienced, from a mere sense of weight and fulness, or vague abdominal discomfort, when the secretion is deficient, to a burning pain when the acid is in excess, or the juice is poured out into an empty stomach. Acid eructations and gaseous expulsions are frequent, and vomiting generally gives relief, though causing a smarting in the gullet from the nature of the ejecta. In hypersecretion and hyperchlorhydria the appetite is often good, and frequently food,

particularly if it be nitrogenous, relieves the pain, by combining with the acid, but in the reverse condition the desire for food is variable, capricious, or wanting. As a rule, the symptoms supervene soon after a meal when the secretions are deficient, and perhaps not for two or three hours when there is excessive acidity.

Owing to the very inefficient digestion of the food when the juice is diminished, the remote digestive troubles such as lassitude, headache, etc., are commonly complained of, and the case corresponds generally to a chronic gastritis, which indeed may be the cause. In excessive secretion, so long as there is food to deal with, symptoms are for the most part in abeyance, since digestion of proteids at least is effected, and the evils of excessive carbohydrate fermentation are averted; but when the stomach is empty, then the superacid fluid acts as an irritant on the mucous membrane, which, there is reason to suppose, is often hyperæsthetic in such cases. As further contributing to the pain that is felt, pyloric spasm is liable to occur, being excited by the over-acidity. Irregularities of the bowels, constipation, or diarrhœa are almost surely present. Regard must also be paid to other manifestations of the underlying neurosis, which may or may not overshadow those referable to the digestive organs.

Very little is known concerning variations in the quality or amount of the pancreatic juice and succus entericus. The alteration which these secretions normally exhibit under different dietetic and other conditions (Pawlow) suggest the probability that they vary in composition and quantity and hence in digestive capability in disease. Complete exclusion of the bile from the intestine is of frequent occurrence, but the interference with the digestive processes consequent thereon is by no means constant, and is often but slight, consistent with the entirely subordinate part taken by the bile in the intestinal changes.

It has been held, and as the writer thinks with good reason, that many cases of so-called "mucous colitis," in which large quantities of mucus are voided with considerable pain and abdominal distress, are properly to be regarded as due to a secretory neurosis which leads to excessive activity of the mucous glands of the colon (p. 287).

Having regard to the marked differences in the digestive process as it is carried out in the stomach and in the intestines, and that, although not indispensable, the gastric changes in the ingesta are an advantageous preliminary to those taking place lower down in the canal, it might be supposed that such differences would be met with in the symptoms of gastric dyspepsia and intestinal indigestion as to allow of a differential diagnosis of those states being readily made.

But this is not so. It is very seldom that any reliable evidence can be adduced in favour of one condition rather than the other, though the real nature of a case may be suspected. In attempting to draw a distinction, the fact that indigestion arising in either stomach or bowels is likely secondarily to be set up in the other should be recollected. For purposes of rational treatment every effort should be made to ascertain the primary fault, and it is hoped that with further knowledge of the morbid alterations in the intestinal fluids the real explanation for many of the symptoms of indigestion may be obtained and a complete diagnosis be effected.

HÆMORRHAGE FROM THE STOMACH AND BOWELS

Excluding those exceptional conditions in which blood finds entrance into the gastro-intestinal tract from some external source, such as when it is swallowed from the nose, mouth, or pharynx, or the rupture of an aneurysm or extension of a vascular growth into the canal; and admitting the possibility of the transudation of blood through intact capillaries (see Vol. II. pp. 310, 311), hæmorrhage from the stomach or intestines may be taken to signify a breach in the continuity of some vessel or vessels. This may be represented by a rupture of capillaries allowing an oozing of but a moderate amount of blood (diffuse), or the bleeding may amount to several pints from the perforation of a vessel of considerable size (focal).

The conditions antecedent to the vascular lesion are much the same for the stomach as for the intestines with certain special features appertaining to each. They may be conveniently grouped in the following manner, it being remembered that any such arrangement is more artificial than natural, and that the cause in any given case is often a complex one :—

(i.) *Trauma.*—Wounds of the gastro-intestinal walls, whether from without, as stabs or crushing, or from within, due to pins, fish bones, or corrosive fluids that have been swallowed, are among the rarer causes of bleeding. Violent retching or vomiting may also cause rupture of small vessels in a normal stomach, and still more likely should there be any ulceration or disease of the vessel walls.

(ii.) *Ulceration and erosion.*—That an ulcer, whether "peptic," typhoid, dysenteric, tuberculous, or of any other nature, should in its progress break into a vessel and lead to hæmorrhage is obvious. The erosion taking place into a vessel in which no previous obliteration has been established and the walls of which are unable to contract, with an increased blood tension in addition, sufficiently explain the

hæmorrhage; and this is the commonest of all causes both in the stomach and intestines, ulceration of a malignant growth being probably the next most frequent. It is seldom, if ever, that any of these conditions exists without some bleeding, much or little, taking place. It may be that the lesion, more particularly in the stomach and first part of the duodenum, is exceedingly minute, requiring the most careful examination for its detection, even injection of the vessels with air or coloured fluid; numerous fatal cases from such are on record. Having regard to the facility with which rupture of the loosely supported capillaries and small vessels of the mucosa may occur, it is quite likely, as has been suggested, that these pore-like erosions of the surface are sometimes really the result of the hæmorrhage, which breaks away the epithelial layer and subjacent tissue. Besides such cases there remains a considerable number in which a severe gastric hæmorrhage occurs, that is seldom fatal, almost invariably in young women between eighteen and twenty-five years, who are chlorotic and very commonly suffering from some menstrual irregularity, such as amenorrhœa, and who are usually very constipated, but in whom, except for mild dyspeptic symptoms, other evidences, such as pain and vomiting, are almost or quite wanting. It has been the practice to reckon such as cases of gastric ulcer upon very insufficient post-mortem evidence, and some at least which does exist has quite negatived the existence of such a lesion, even of the minutest character. Thus the etiological and clinical differences, which will be further considered (p. 297), between these and true gastric ulcer, as well as the observed anatomical distinctions, show that they should be regarded as in a separate category. A satisfactory explanation, however, of the bleeding, which may be very profuse, is still wanting. That it is not due to ulceration in the proper sense of the term seems certain—at least in many cases—for when the stomach has been opened during life, an oozing has been observed from minute bleeding points, quite unlike an ordinary ulcer, and the cause is probably far from simple. The frequent association with chlorosis suggests that this morbid state of blood and vessels favours hæmorrhage, as it is well known many blood states do; the connection with amenorrhœa and the frequently recognised time relation of the bleeding to the menstrual epoch, which has given rise to the older view that such bleedings were "vicarious menstruation," further suggests a causal relationship between the events; and, lastly, the excessive hydrochloric acid secretion which is often to be found in such subjects very likely leads to a hyperæmia which also favours the escape of blood. Blood may pass into the stomach and be

vomited, from a duodenal ulcer the distinguishing characters of which are described on p. 301. Vascular polypi which are occasionally found in the lower bowel and rectum may bleed from abrasion of their surface by the fæces or from over-engorgement.

(iii.) *Hyperæmia and congestion.*—Reference has just been made to the possibility of gastric bleeding being the result of an active hyperæmia, consequent on the irritation of excessive acid secretion. To the same cause may be attributed the so-called "vicarious menstruation," an unfortunate term, since "the bleeding is in no sense 'substitutive.' The disturbance has not changed its centre, but extended its range—it may be to the stomach, or nose, or bronchial tract. The bleeding may occur in conjunction with the menstrual flow, or when there is amenorrhœa. And there is nothing very extraordinary in its existence, when one recalls the changes that the time of 'heat' will produce in animals, *e.g.* in the plumage of birds, and the widespread disturbance, physical and mental, set up by menstruation in women. No doubt, however, some of the cases of gastric bleeding at the menstrual period are due to ulcers, which, owing to the hyperæmia of the organ, have a natural proneness to bleed at that time."[1] To an active determination of blood to the gastric mucosa may perhaps be ascribed the hæmorrhage occasionally met with in pneumonia, and that which sometimes follows on operations. As a phase of the inflammatory state, hyperæmia determines the bleeding, usually very slight, which occurs in the course of a gastritis or enteritis.

Next in order of frequency to ulceration as a cause of gastro-intestinal hæmorrhage comes mechanical congestion of the gastric and mesenteric vessels. This is most commonly due to obstruction of the portal circulation, either from cirrhosis of the liver, or from obstructive heart disease, the latter being much rarer than commonly supposed. The cause of the bleeding in cases of this kind has generally been explained by rupture of the over-distended capillaries and smallest venules, and, no doubt, such is sometimes the explanation. More often, however, it is due to the rupture of varices which are developed at the junction of the œsophageal and coronary veins of the stomach at the lower end of the gullet. The communication between the systemic and portal vessels in this situation is very free and direct, and being intrathoracic is subject to the aspirating effects of inspiration (Vol. IV. p. 312); an obstruction, therefore, in the hepatic circulation, especially if the distensibility of the liver be diminished, as it is in cirrhosis, tends to distend these channels as,

[1] Dr. Dawson on Hæmatemesis, *Tr. Med. Soc. Lond.* 1902-3.

in a lesser degree, it does at the junction of the hæmorrhoidal and systemic veins, leading in both situations to the formation of varices or piles. The point of rupture of the œsophageal lesion may be exceedingly minute, and require most careful looking for after death ; sometimes, though rarely, definite ulceration is to be found. Although these œsophageal varices are usually produced under the circumstances described, it is nevertheless certain that they are occasionally met with in a perfectly normal circulation, as rectal piles most frequently are, and are then to be regarded as due to primary affections of the vessels.

Limited areas of congestion of the gastro-intestinal tract may be responsible for profuse bleeding, such as from thrombosis or embolism either of the mesenteric vessels, or of their main branches (see p. 545). It may be observed that when bleeding from the stomach or bowels follows on obstructive heart disease, thrombosis, in addition to the venous congestion, presumably exists. The venous engorgement, consequent upon an intussusception of the bowel, may lead to the same. The attacks of gastric bleeding that often complicate cases of splenic anæmia (Vol. II. p. 353), and that cease after removal of the spleen, have been sometimes supposed to be due to obstruction to the return of blood by the vasa brevia from the cardiac end of the stomach into the splenic vein, these being pressed upon by the much enlarged organ.

Bleeding from the stomach and bowels has long been known to accompany, with considerable frequency, that curious vascular condition known as "pulsating aorta" (see p. 523). In a series of cases published by Dr. S. Phillips (*Brit. Med. Jour.*, 1887, 8th Oct.), no ulceration was to be found, and no cause could be assigned. It may be that, like the aortic state itself, some neurosis may underlie the phenomenon, a vasomotor paralysis leading to over-fulness of the vessels. This perhaps also explains the post-operation hæmatemesis already referred to, occurring as this does about three days after operation, and to be compared to the suppression of urine which is sometimes seen under like circumstances. Death from this cause is not unknown. It is possible that this nervous element has some share in producing the hæmorrhage in the chlorotic young women already described as of complex origin.

(iv.) *Changes in the vessel walls.*—Degenerations of the coats of the vessels, such as may be met with in lardaceous disease, and Bright's disease, leading occasionally to the formation of miliary aneurysms, are among the rare causes of bleeding from the stomach and bowels, sometimes apparently without the existence of any

definite ulceration, though the impaired vitality of the mucosa supplied by such vessels probably allows in the stomach of the destructive action of the gastric juice.

(v.) *Morbid blood states.*—Hæmorrhage from the mucous membrane of the stomach and bowels, as from other surfaces, is a frequent symptom of such diseases as purpura, scurvy, leukæmia, splenic anæmia, yellow fever, acute yellow atrophy of the liver, malaria, malignant endocarditis, and other infectious diseases, such as scarlet fever and diphtheria. Besides the profound alteration in the blood which characterises these and similar maladies favouring its escape from the vessels, there is good reason to suppose that there are coincident if not consequential changes in the vessel walls which facilitate their rupture (Vol. II. p. 311). The association of urticaria with gastro-intestinal bleeding has been noticed, and both are probably of toxic origin. Dr. Dawson (*loc. cit.*) has suggested that the excretion of irritant substances by the stomach in some toxæmias may thus cause bleeding, and is disposed thus to account for the hæmatemesis that has been observed in severe cases of appendicitis.

(vi.) *Gastro-intestinal hæmorrhage in the new-born.*—In a small proportion of infants bleeding in this situation may occur, commonly showing itself within six hours, though occasionally not for five or six days. Its causation is uncertain. The sudden and violent disturbance of the abdominal circulation which takes place on ligature of the umbilical cord may predispose to it, though it can scarcely be the sole cause. Some have referred it to emboli of infective origin, and very rarely gastric or intestinal ulceration has been found. It is not infrequently fatal.

The **effects** of gastric or intestinal hæmorrhage will largely depend upon the amount of blood lost. When the quantity is exceedingly small no symptoms whatever may be experienced. Repeated small losses, however, may establish a condition of anæmia and its consequences. The general effect on the patient of an appreciable bleeding has been fully described in Vol. II. p. 361, to which account may be added the statement that after profuse hæmorrhage from the stomach, certain disturbances of sight are more likely—though very rarely—to follow, than when the loss of blood occurs in other situations; that this is so is probably connected with the straining that accompanies the vomiting. Besides a transient amaurosis and amblyopia, a permanent blindness has been known to set in. It may also be observed that a severe gastric hæmorrhage is particularly prone to cause faintness, much more so apparently than when a similar loss of blood takes place from the

lungs—a point of some clinical importance. A sensation as of a warm fluid flowing into the abdomen is occasionally complained of, but otherwise intestinal hæmorrhage is seldom possessed of characteristic features. Abdominal pain may accompany it, and is specially severe in embolism of the mesenteric arteries, but this symptom is not to be attributed to the bleeding, both being associated manifestations of a common cause. The occurrence of these indications in the course of a disease liable to lead to this condition would point to hæmorrhage, especially if there be a fall in temperature from a previous pyrexial state.

When any considerable escape takes place into stomach or bowels, the blood is usually vomited (hæmatemesis) or passed per anum, or both. Sometimes, however, an intestinal hæmorrhage may prove fatal without any being voided, and a period of two to four hours generally elapses between the actual escape from the vessel and its appearance externally. Not so in the stomach, where vomiting occurs almost at once—fresh blood being very provocative of emesis—unless there have been a slow oozing, which apparently might continue for some time before it is rejected. Previous to the hæmatemesis the patient is apt to feel sick and to become pale and faint.

The quantity of blood lost varies considerably from a mere trace up to several pints. As regards the amount brought up from the stomach, an admixture with gastric secretion or fluid previously swallowed may help to make the quantity appear greater than it really is, and the exaggeration of patients and their friends in this respect is not to be forgotten.

More important is the appearance of the vomit or the stool. The blood may be quite bright or of a darker colour when brought up, and clot on standing. Such may be presumed to have come from a vessel of some size, whether artery or vein, and not to have remained any time in the stomach. When, however, it is of a brownish colour and of a coffee-ground appearance, and does not coagulate, it has almost certainly been effused slowly, and has undergone change by the gastric juice, the hæmoglobin having been decomposed into hæmatin. Such material is commonly seen with gastric cancer, and sometimes in the passive congestion due to cirrhosis of the liver. But these distinctions are not absolute as between focal and diffuse bleeding, the time the blood has remained in the stomach after escaping from the vessels being the chief cause of the differences, rather than the nature of the lesion. The appearances of blood when passed by the bowel vary considerably. The

position of its source, the quantity, and the duration of its stay in the canal, largely determine the extent of change in the blood, and its degree of admixture with the fæces. If the cause be situated immediately within the anus, or the blood be sufficient in amount to escape alteration, then the red colour is retained. The hæmatin is readily affected by the sulphuretted hydrogen in the canal, and converted into a blackened material, sulphide of iron being formed, which stains the fæces; or a black tarry substance is evacuated, being the altered clotted blood (*melæna*). When the blood has undergone this change, the source of it is in the stomach or first part of the duodenum; blood from the colon—where it is usually due to ulceration—is generally passed adherent to the fæces.

The **diagnosis** of the source of the blood that is vomited or voided per anum is often as difficult as it is important both for treatment and prognosis. Dealing first with hæmatemesis, it is essential at the outset to make sure that blood does come from the stomach and not from the air passages. The characters of hæmoptysis are fully described in Vol. IV. p. 108, and the chief points of distinction may be briefly recapitulated. In hæmatemesis the blood is apt to suddenly well up into the mouth without any previous coughing, or may be a single cough, or cough may follow the vomiting, and perhaps nausea; it is bright or dark, acid, and rarely frothy, comes up in quantity and at once, ceasing abruptly, and not often repeated, except at considerable intervals; sooner or later it is followed by melæna. In hæmoptysis, though the blood may escape from the mouth even more suddenly than when it comes from the stomach, it is almost always preceded and accompanied by coughing, and subsides gradually over some period; the blood is alkaline, more or less mixed with air, and a blood-stained mucus is expectorated for some time after the chief bleeding has ceased; and the bleeding is less likely to induce syncope. Sometimes the distinction is not so easily made as might be supposed from this *résumé* of the several characters, especially if it depend on the account of a third person.

Assuming it to be determined that the blood has come from the stomach, the diagnosis of its origin is to be based upon the consideration of (*a*), the recognised causes of gastric hæmorrhage as previously set forth; (*b*), the age, sex, and condition of the patient; (*c*), the presence of easily ascertained disease of the heart and liver; (*d*), the other symptoms of gastric disease, such as pain, vomiting; (*e*), the previous history of the patient and the onset of the attack; and (*f*), the quantity and appearance of the blood brought up, to

which only a very partial value is to be attached. In this connection the fact that blood from the nose, pharynx, or mouth may be swallowed and afterwards vomited must not be forgotten, nor the fictitious hæmatemesis of hysterical women and malingerers. Similar considerations should underlie the investigation of the cause of an intestinal hæmorrhage, but here somewhat more importance may be attached to the appearance of the stools.

Prognosis.—It is not often that hæmorrhage from stomach or bowels is immediately fatal, though it may be so should an aneurysm have burst into the canal, and very exceptionally bleeding from an ulcer in the stomach may be fatal, a result which is commoner in a typhoid lesion in the intestine. Now and then also death follows soon upon bleeding due to cirrhosis of the liver. Even repeated bleedings may be recovered from, though if the recurrences be long continued ill effects of necessity follow, but the cause of the hæmorrhage, rather than the actual loss of blood, chiefly influences the ultimate result. The great probability of complete recovery even after severe bleeding, and in those cases occurring in chlorotic girls, offers a strong contrast to the hæmatemesis which takes place in true gastric ulcer, with its risk of death from bleeding, perforation, subphrenic abscess, or other complications. The realisation of this fact is of great importance in respect to the question of operative treatment, which may be as justifiable in the latter case as it is inadmissible in the former; and, as already said, no reliable assistance, unfortunately, in the diagnosis of these two forms of hæmatemesis—diffuse and focal—is to be obtained from the appearance of the vomited blood.

PERFORATION OF THE STOMACH OR INTESTINES

Perforation of the gastro-intestinal tract usually occurs as the result of ulceration, of which it is a not infrequent complication. It is estimated that the accident happens in about 6 per cent of all gastric ulcers, and in a somewhat less proportion in intestinal ulceration. Those ulcers which are the most acute in their course are those most likely to perforate, hence the peptic ulcer of the stomach and duodenum, and the typhoid and acute dysenteric ulceration of the bowel are those in which the complication most commonly occurs. When the normal course of the ulcerative process is slower and progressive, inflammatory tissue is likely to be developed in front of the destructive change, thus forming adhesions which in most cases prevent actual perforation, though the entire thickness of the bowel may be destroyed. Such ulcers are the tuberculous and the

several forms of chronic ulceration of the colon; though perforation sometimes happens even in these affections. Sometimes more than one perforation occurs, even in the stomach, though more frequently in the intestine. Among exceptional causes of rupture of the canal may be mentioned extreme flatulent distension above an obstruction with degeneration of the wall of the tube; destruction by corrosive fluids, or sharp objects, pins, nails, spicules of bone that have been swallowed; ulceration into the lumen of the bowel from without, as by a calculus from the gall-bladder, though in such cases communication with the peritoneum is generally prevented by the formation of adhesions; injuries and wounds, gunshot and other.

The consequences, according to the situation of the perforation, are either communication with the greater or lesser peritoneal cavity and escape of the gastro-intestinal contents into these sacs; or into the post-peritoneal connective tissue, setting up abscess; or into another section of the canal, adhesions between the contiguous portions having been previously established.

The last-named condition is not necessarily accompanied by any symptoms, although, owing to communication for instance between the stomach and colon, the acid contents of the former are likely to cause diarrhœa and give a distinctive character to the motions or to the vomit, but the actual perforation probably causes no symptoms. It is far different, however, when the ulcer opens direct into the peritoneal cavity, for then severe and urgent manifestations appear, marked usually by extreme suddenness of onset. The most constant of these is violent pain, which is regarded as marking the actual perforation; it is frequently situated at first over the site of the lesion at the epigastrium or in the region of the cæcum, but it is sometimes referred to the umbilicus, especially if it be the intestine that have perforated. Too much dependence, however, must not be placed upon the locality of the pain as indicating the situation of the lesion, as a gastric or duodenal perforation has on several occasions been known to occur when the pain was over the cæcum. The pain is sometimes followed by vomiting, oftener perhaps in the case of gastric perforation, and occasionally associated with rigors. When the condition is one of fever, as in typhoid, it is usual for the temperature to fall even below the normal, subsequently rising, but this is not invariable, and even an increase has been known. General abdominal tenderness is soon complained of, and the belly becomes tumid and distended from the escape of air into the peritoneal cavity, the hepatic dulness sometimes becoming lost in

consequence, though the frequency and hence the significance of this sign is far from being what was at one time supposed; the abdomen continues to move in respiration, the immobility and rigidity of the walls being a later phenomenon. It has been sought to distinguish the condition here described from simple colic by the attitude of the patient and the relief afforded to the latter by doubling the trunk on the thighs and maintaining pressure over the abdomen; this, however, is not constant, and the writer has recorded a case of perforation of the duodenum, as verified post-mortem, which was mistaken for colic for this very reason. The patient's expression is anxious and the features are drawn, often with pallor of face and cold sweats, but consciousness remains. The pulse is quickened and respiration a little hurried. These symptoms continue until after about six hours, when those more distinctive of peritonitis set in (p. 490). It is very rare for death to take place at this stage. It is obviously important, with a view to operative treatment, to ascertain whether it be the stomach or duodenum or the appendix that has perforated. This is often impossible, especially if there have been no previous symptoms, as is sometimes the case. The history becomes valuable when it is obtainable, and in such maladies as typhoid fever and dysentery the situation of the perforation is at once suggested, although, should the patient be comatose, the complication may be overlooked. The fact that gastro-intestinal ulceration may be altogether latent as regards symptoms until perforation occurs should be remembered.

OBSTRUCTION OF THE GASTRO-INTESTINAL CANAL

Many morbid changes both within and without the stomach and bowels may cause obstruction to the onward progress of the contents and give rise to symptoms dependent on this condition.

In the STOMACH extensive cicatrices, especially when involving both surfaces of the organ, may lead to obstruction; but the pylorus is the most frequent situation for stenosis, the causes of which have been enumerated at p. 188.

INTESTINAL obstruction is due to a large variety of conditions which affect different portions of the canal in different degree. They may be grouped thus [1]:—

1. *Strangulation by bands or through apertures.*—Such bands

[1] The arrangement here followed is based upon that adopted by Sir F. Treves, Bart., to whose excellent article on the subject in Allbutt's *System of Medicine*, vol. iii., the writer is much indebted for this section.

may be the result of peritonitis, of abnormal attachments of normal structures, as the appendix, or of abnormal diverticula, Meckel's, and others. The apertures may be slits in the mesentery, the foramen of Winslow, peritoneal fossæ, or in the diaphragm, giving rise to internal hernia, or the abdominal rings producing external hernia.

2. *Volvulus*,[1] including twisting of the bowel on its mesenteric axis, and the intertwining of two coils of intestine sufficient to cause obstruction. It is clear that only such sections of the tube as are not bound down to the parietes may be thus affected, viz. jejunum, ileum, ascending colon and cæcum, and sigmoid colon. The latter is by far the most frequent situation, it being brought about by chronic constipation, enormous dilatation of this portion of the gut being the result.

3. *Intussusception* or the invagination of one portion of bowel with its mesentery within another may occur in the jejunum and ileum, in the sigmoid colon and upper part of rectum, or the lower part of the ileum may pass into the large intestine, in such a manner as either to carry with it the cæcum and ileo-colic valve, the latter forming the "apex" of the intussusception, which may protrude at the anus (ileo-cæcal), or be inverted into the colon through the valve which remains *in situ* (ileo-colic). Of all causes of fatal intestinal obstruction this may be regarded as the commonest, the most frequent form being the ileo-cæcal and next to that the jejunal, the ileo-colic being the rarest. The invaginated portion, consisting of an "entering" and of a "returning" layer, is almost always received into the adjoining section of bowel ("sheath") in front, *i.e.* towards the anus; in dying persons, however, especially in children, the intussusception may be in the reverse or upward direction, and are then frequently multiple. By this arrangement the mucous surfaces are always in apposition, and the serous surfaces the same.

The condition, which appears immediately to depend upon some irregular peristalsis of one part of the bowel with inaction of another part, is found in connection with violent purging, worms, intestinal polypi, and sometimes after straining or excessive movements of the body; oftener, however, nothing abnormal can be ascertained, and it is very doubtful how far the above-mentioned conditions are really effective in producing the lesion.

When once an intussusception is formed, if it increase in extent, it usually does so at the expense of the outer or receiving layer, though this is not so in the ileo-colic variety, at least at first. Very

[1] The term ileus sometimes applied to any form of intestinal obstruction is more properly applicable to this variety.

rarely a primary intussusception may be invaginated into a secondary one, and then again even into a third. The ileo-cæcal and ileo-colic forms involve the largest portions of bowel; those of the small intestine rarely exceed 6 or 8 inches, and those of the colon are much shorter still. The disposition of the mesentery in the

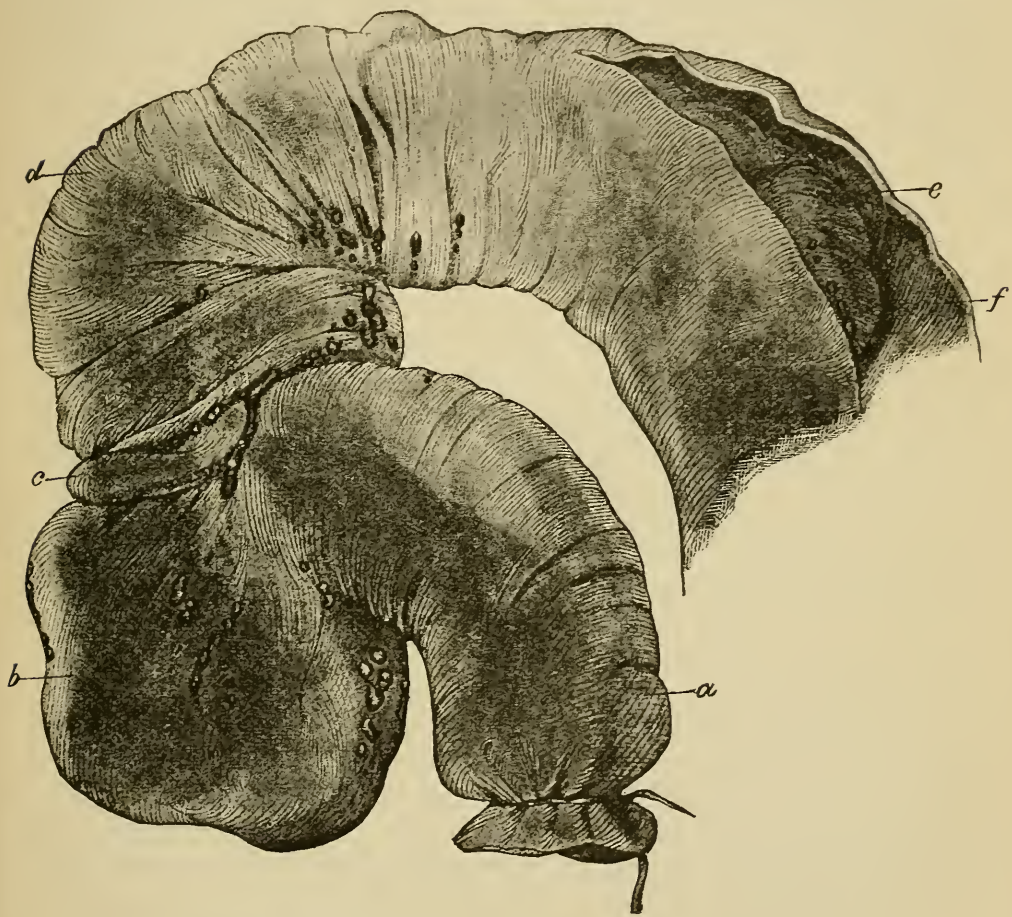

FIG. 9.—Intussusception of intestine at ileo-colic valve. *a*, ileum; *b*, cæcum; *c*, vermiform appendix; *d*, transverse colon; *e*, invaginated intestine. (Ziegler.)

intussusception, and the traction which it exercises, give to the invagination a curvature. This circumstance, together with the obvious liability of the parts concerned to become congested and œdematous from the compression to which they are subjected, causes a more or less complete obstruction of the canal, and a bleeding into its lumen. If this become excessive the invaginated portions become gangrenous, and may separate and be voided, some-

times entire, the continuity of the bowel being maintained by adhesions. Neither obstruction nor gangrene, however, are invariable, and differences exist in the degree to which they are manifest in the acute and chronic cases.

4. *Stricture.*—By this is meant constriction of the gut by (*a*) cicatrices of previous ulceration or gangrene, (*b*) infiltration of the walls by new growths—commonly malignant, and (*c*) congenital narrowing. The character of these lesions has been previously described. The stricture may be single, as it usually is when in the large intestine, or multiple, as it sometimes is in the small bowel, and in some congenital cases. The colon is much oftener the seat of obstruction of this nature than the small intestine, whether cicatricial or neoplastic, and the latter cause is by far the commoner.

5. *Obstruction from conditions within the bowel.*—These are of various kinds :—

(*a*) New growths of a benign character and usually pedunculated, single, multiple, and varying in size from a pea to a pear. They often give rise to no evidence of their existence until post-mortem examination, but at times may be responsible for obstruction, occasionally by causing intussusception. Their characters have been described on p. 184.

(*b*) Foreign bodies which have been swallowed, aggregations of indigestible vegetable fibres, seeds and husks, enteroliths, or gall-stones which have ulcerated through from the gall-bladder into the bowel. Examples of the last-named have been met with measuring 3 to 4 inches and more in circumference.

(*c*) Fæcal impaction. This is probably the commonest cause of obstruction, though not nearly so often causing death, for which intussusception, strangulation by bands or through apertures, stricture, and growths of the bowel are most frequently responsible, and in that order. Volvulus and pressure on the intestine by growths outside are rarer.

6. *Pressure on the bowel from without.*—Abdominal tumours, especially those in the pelvis, a gravid uterus, or a displaced kidney, may so press on the intestine as to offer a partial obstruction to the progress of its contents. The rectum and sigmoid colon are the parts most likely to be affected. Extensive adhesions between the intestines, the result of chronic peritonitis, will also interfere with the movements of the bowels and so far cause obstruction, though the lumen may be perfectly patent.

7. Besides the above-mentioned varieties of what may be termed mechanical obstruction, a *functional obstruction* will result from

impaired propulsive power in the gastro-intestinal musculature, such as follows on atony and dilatation, or violent interferences with the circulation, such as thrombosis of the superior mesenteric vein.

Considerable difference is met with in the sex and age incidence of these various forms of intestinal obstruction. "Strangulation by bands and through apertures is a little more common in males than in females, is met with mostly in young adults, and is rare after forty. It is still rarer before ten. Internal herniæ have been shown to be more common in males, and the greater number of the recorded cases have fallen between the ages of twenty-five and forty-five. Volvulus of the sigmoid flexure is about four times more common in men than in women. It is most common between forty and sixty, and indeed is rare before forty. Acute intussusception is a little more common in the male sex. It mostly attacks the young; 50 per cent of the cases are under ten years. Strictures are equally common in the two sexes. The non-malignant occur about early middle life; the cancerous stricture is rare before forty. Certain strictures of the intestine are congenital. Obstruction, due to tumours external to the bowel, is obviously more common in women and in adults. Obstruction due to impacted gall-stones is commoner in women than in men, and the average age falls between fifty and sixty-five. Fæcal accumulation occurs with greater frequency in women than in men. It is most common in adults and in the aged, and is not infrequent in the insane and hysterical" (Treves, *loc. cit.*).

Clinical characters and course.—A consideration of the numerous varieties of intestinal obstruction will show that they readily fall into acute and chronic forms, with acute or gradual onset, and a rapid or lingering course towards death or recovery. Not infrequently one of long duration terminates in the supervention of an acute condition. The chief forms of acute obstruction are strangulation by bands or through apertures, volvulus, acute intussusception, and acute blocking by foreign bodies; whilst the more chronic obstructions are represented by stricture, chronic intussusception, and fæcal accumulation.

The distinctive characters of these two groups are conveniently represented in the following table:—

	ACUTE INTESTINAL OBSTRUCTION	CHRONIC INTESTINAL OBSTRUCTION
Onset	Sudden; usually without apparent exciting cause, sometimes after severe exertion or constipation. Initial rigor rare.	Gradual.
Pain	Very severe; continuous with paroxysmal exacerbations. Usually referred to umbilicus, sometimes to site of obstruction, hence unreliable for diagnosis. Due to injury to bowel, to colic, and later to peritonitis.	Colicky attacks of moderate intensity, with intervals of freedom, increasing in frequency, severity, and duration. The intermittence in contrast to the constancy of the pain characterises partial as opposed to complete obstruction. May be relieved by pressure, but often excited by food, purgatives, or enemata from the induced peristalsis. Relief often follows action of bowels when small bowel involved.
Tenderness	Slight at first, and limited to seat of lesion; may increase and become considerable and diffuse later from peritonitis.	Very variable in degree.
Vomiting	Sets in early; at first of gastric contents, then bilious, becoming feculent in about five days in small bowel obstruction, and later in colon occlusion. Continues with retching, the most distressing symptom throughout, and gives no relief. At first reflex, afterwards due to reversed axial current of the intestinal contents.	Slight as a rule throughout, often induced by food; rarely fæcal. Varies much in degree, but less marked in large than small bowel obstruction. Nausea and anorexia may persist.
Constipation	Usually absolute from first, even to passage of flatus, and when obstruction is high up, being then probably due to reflex inhibition of peristalsis. More or less *diarrhœa*, with muco-sanguineous stools, may continue in intussusception.	Moderate at first, and relievable; increases, and is often interrupted by attacks of "spurious diarrhœa" induced by irritation of retained fæces, and most marked when obstruction is low down in colon. Finally becomes absolute, and of days' or weeks' duration.
Abdomen	Normal, tumid, or distended. Meteorism, which may be only slight, most marked in colon obstruction, and specially sudden and severe in volvulus of sigmoid. Rigidity in later stages only, or absent throughout. Definite tumour rarely detected, except an intussusception, especially the ileo-cæcal and colic varieties, frequently also felt in rectum.	Degree of distension variable, may be wanting, and abdomen even retracted. Often visible and painful peristalsis, with gurgling and rumbling. Outline of distended colon or of intestinal coils may be apparent. Tumour may be felt if there be a growth or fæcal masses.
Tongue	Very coated, later dry and brown.	White and coated. Breath offensive, and at last fæcal.
Thirst	Intense, and proportioned to the vomiting.	Not special.

	ACUTE INTESTINAL OBSTRUCTION	CHRONIC INTESTINAL OBSTRUCTION
Tenesmus	Only met with in intussusception.	Chiefly in malignant stricture of lower part of colon, and often accompanied by hæmorrhoids.
Collapse	Occurs early. Determined in degree by suddenness of strangulation and extent of gut involved; is more marked the nearer the lesion is to the stomach.	Absent.
Temperature	Subnormal; sometimes slightly raised with onset of peritonitis.	Normal.
Pulse	Small, rapid, and thready.	Variable, but progressively quick and feeble.
Urine	Diminished up to suppression, very much in proportion to the acuteness of the attack, the degree of collapse, and severity of the pain. Bears no relation to site of the obstruction as formerly supposed.	Nothing special.
Respiration	Thoracic, shallow, and, if much abdominal distension, is embarrassed.	No special character, unless great distension.
Hiccough	Occasionally, and may be very severe.	Very rare.
Cramps	Have been noticed in acute strangulation.	Not observed.
Consciousness	Generally retained to end; stupor and coma in final toxæmia. May be delirium.	Retained throughout, or an increasing coma towards the end.
Expression	Anxious, features drawn and pinched, eyes sunken, voice weak, pallor with cold sweats.	As in acute, but more gradually developed.
Progress	If unrelieved, symptoms quickly increase and exhaustion comes on.	Irregularity in severity of symptoms throughout; intervals of feeling fairly well. Becomes progressively weaker and emaciated, and generally worse as symptoms are more continuous and severe. Average duration about six months.
Death	In 5 to 7 days from septic absorption; exceptionally within 24 hours. Often preceded by subsidence of pain, vomiting, and other symptoms as toxæmia develops and sensibility is blunted, and sometimes by a copious motion.	From perforation or other complication, but oftener from exhaustion due to pain, vomiting, and inability to take food and septic absorption. Sometimes as the acute preceded by a large evacuation.

In thus contrasting the symptoms of acute and chronic intestinal obstruction it should be realised that in the former pain and vomiting are the most prominent manifestations, the constipation, however complete, being scarcely if at all complained of. But in the chronic condition it is the constipation that becomes from the first more and

more distressing, other symptoms, as a rule, being less marked, though liable to progressive increase in severity.

In attempting to ascertain whether the obstruction be situated in the large or in the small bowel, a proceeding which is often far from easy, the following points should be kept in view:—(i.) The probabilities conferred by the sex and age of the patient as previously set forth; (ii.) the greater frequency with which the large gut is obstructed, reckoning all causes; (iii.) the more sudden onset, more acute course, and generally the greater severity of the symptoms, both local and general, when the small intestine is the seat of the obstruction, although volvulus of the sigmoid is very acute in character; and (iv.) the greater frequency of stercoraceous vomiting and of suppression of urine in small bowel obstruction. Much less reliable assistance is to be obtained from the condition of the rectum on digital examination, unless of course any obstruction be felt within range of the finger. An empty dilated state of this part of the bowel, the walls feeling quite smooth, known as "ballooning," is often associated with stricture of the colon, but not invariably so. It appears to be of the nature of a paralytic distension, and disappears under an anæsthetic. Sometimes the degree and distribution of the meteorism may help, being much greater when the colon is concerned, the outline of the bowel being sometimes clearly marked out, and is most marked and severe in volvulus of the sigmoid. The upper zone of the belly may for a time be alone distended when the jejunum is occluded, or the central parts only when the obstruction is at the lower part of the ileum, but the distension tends to become uniform, and too much reliance must not be placed upon these features. It may be noted that neither vomiting nor diarrhœa has much effect in reducing the tympanites. The attempted diagnosis of the seat of the trouble by a long tube passed into the bowel, or by the quantity of fluid that may be injected per anum, is quite unreliable. The same applies to an examination of the stools as regards their consistency, shape, and calibre, unless the difficulty be at or just within the anus, when narrow, flattened, grooved, or "pipe-stem" motions may be voided. Otherwise it cannot be too strongly insisted on that stools perfectly normal in appearance may be passed, although a stricture should exist even as low down as the sigmoid flexure, the fæces being then moulded to the usual size and shape in the rectum. The presence of blood should be noted as well as its colour and appearance; it may occur in connection with malignant growths or with intussusception, but otherwise is very unusual from intestinal obstruction.

Inasmuch as many of the conditions which cause obstruction, whether acute or chronic, are likely to lead to peritonitis, the symptoms of this affection are liable to supervene on those determined by the original lesion. At the same time, however, it should be remembered that acute peritonitis bears a considerable resemblance to acute obstruction ; the greater degree of pyrexia, and of tenderness, with less severe vomiting, which is never fæcal, and only partial constipation, are features that should distinguish the peritoneal affection ; although an intense peritonitis may exist with a subnormal temperature. Many of the symptoms of obstruction are also simulated by the condition known as "phantom tumour," by acute pancreatitis, and sometimes even by torsion of a floating kidney, or by an impacted calculus in the ureter.

Nor should it be forgotten that the administration of opium modifies or obscures the symptoms, some of which, such as the pain, vomiting, and suppression of urine, are considerably bettered, leading to a false appearance of improvement ; the tympanites, however, will probably increase.

When the obstruction is due to a malignant growth the emaciation and weakness will probably proceed more rapidly than when a merely mechanical difficulty exists.

W. H. Allchin.

The Physical Examination of the Stomach and Intestines

The stomach is liable to abnormalities of shape, position, and size.

As regards *position*, there may be displacement in an upward, lateral, or downward direction, though the latter is alone of clinical importance. In downward dislocation (gastroptosis) the lesser curvature is no longer under cover of the liver, the pylorus is not under the right costal margin, and no portion of the organ occupies the epigastrium. The viscus is not prolapsed in its entirety owing to the greater fixity of the cardiac end. The extent and variety of the displacement are determined by the position of the pylorus and the lesser curvature. Without a knowledge of these the position of the greater curvature is no criterion of prolapse, for it might equally indicate increased size. The lesser curvature may be vertical and the organ occupy the left half of the abdomen, or it may have a more or less oblique direction downwards and to the right. Frequently these facts can only be brought out by inflating the stomach with gas, when the organ becomes bulged, the epigastrium sunken, and the concavity of the lesser curvature apparent.

Size.—The stomach may be too large or too small, though neither condition need give rise to clinical manifestations. If, however, the organ is not only enlarged, but its motor power be insufficient to efficiently empty the contents, the condition is styled dilatation or gastroectasis. Enlargement of the stomach conjointly with retention of its contents beyond the proper limits of digestion (seven hours at most) are the essential features of dilatation. This residual retention may be the result of pyloric stenosis or impaired mobility, or a combination of both conditions.

In gastroectasis the enlargement of the organ is in all directions, though it chiefly manifests itself clinically by the altered position of the greater curvature. Under normal conditions the greater curvature, even soon after a meal, should not be lower than a point one inch above the umbilicus, and the lesser curvature should always be under cover of the liver. If the lesser curvature be in its proper position and the greater be displaced downwards, the stomach is enlarged. If the lesser curvature be displaced downwards and the distance between it and the greater does not exceed four inches, the condition is one of gastroptosis. The two conditions of dilatation and prolapse often coexist, and the presence of one favours the development of the other. The weight of the overloaded stomach in gastroectasis drags on the attachment, and there are few cases of dilated stomach of long standing in which there is not more or less prolapse. Gastroptosis, on the other hand, may, though less commonly, lead to dilatation.

To form a judgment on the nature of a stomach disorder we need to know, on the one hand, the present symptoms and preceding history, and to investigate, on the other hand, the objective manifestations. Under the latter are comprised the position of the viscus, its size under varying phases of digestion, its motor power, secretory efficiency, and the existence of superficial or deep tenderness, which are ascertained by the following methods of physical and chemical examination.

Inspection.—The most favourable position is for the patient to be on the back on a flat, rigid mattress, with the legs straight, hands by the side, and one pillow only underneath the head. The head of the bed should be against the wall, and just to the left and above the level of the head of the bed should be a window or other source of light.

The shape of the thorax should be noted, especially of its lower part. In gastroptosis it is often long with very little space between the ribs and the iliac crest, and its lower part may show signs of constriction by corsets. The tenth rib, too, is frequently said to be floating where the stomach and other viscera are prolapsed, though this would be better felt than seen. The shape of the abdominal wall is important; there may be hollowing in one place and bulging in another. The contour of a dilated or prolapsed stomach may often be detected if the viscus be sufficiently distended. If there be dilatation the outline of the greater curvature will be visible; if there be prolapse the bulging will be more obvious, the epigastrium will be hollowed, and the concavity of the lesser curvature apparent. A stomach which is both dilated and prolapsed may bulge the whole abdomen except the epigastric region. If the direction of the light be favourable, and the eye of the observer be brought down to the level of the abdomen, the thin outline of the greater curvature and its movements with respiration can sometimes be seen, and in this way the accurate localisation of the stomach is aided.

The pylorus can only be seen if it be the seat of a tumour, and then only in a small proportion of cases.

There is nothing which renders the determination of dilatation and prolapse so easy and definite as the recognition of peristaltic waves travelling across the viscus; these bring out the outline of the organ with great prominence. If these peristaltic waves be very strong, the probabilities of pyloric stenosis being the cause of the dilatation are increased.

Palpation.—The observer should be on the right side of the patient, and his forearm be nearly parallel with the abdomen; a good way is to sit on the edge of the bed facing the patient. The hand should be warm and its movements gentle. Palpation should be methodical, and needless fingering of the abdomen should be avoided as it only serves to make the wall harden. The abdomen should not be prodded with the tips of the fingers and with the hand high above the level of the patient. At first the whole hand should be used and applied flatly, and

later this may be supplemented by palpation with the fingers only. The patient should be asked to breathe naturally, not deeply. There is no advantage in having the knees flexed.

The thickness and integrity of the abdominal wall should first be noted. Sometimes the recti muscles are separated, and the feel of the abdominal wall thereby deceptively modified. Separation of the recti may be brought out by asking the patient when in the recumbent position to make the effort to sit up with the arms folded across the chest. A weak abdominal wall plays an important part in the causation of gastroptosis.

The stomach when partially or wholly full has a characteristic feel of resistance on palpation. When both its size and position are normal these can only be felt after a full meal, and then only imperfectly, and not at all if the subject be stout or the abdominal wall rigid. But if the organ be enlarged or prolapsed this characteristic resistance is often very definite. The convexity of the greater curvature and the concavity of the lesser may often be felt, and are signs of value. The demarcation of the lesser curvature is conclusive evidence of prolapse, and with absence of the latter condition the detection of the greater curvature below the umbilicus is conclusive evidence of enlargement.

Splashing.—This exactly resembles the sound produced by sharply palpating a water pillow, and is produced by like conditions. To elicit the sign, which can be both heard and felt, fluid and gas must be present in the stomach, and the fingers of both hands should be over the stomach area, those of one hand dipping quickly and lightly, while those of the other are at rest, or the stethoscope may be used in place of the hand at rest. Splashing is not *per se* a morbid sign, for it may quite properly be present in a healthy person after a full meal. It is only of importance when present in the wrong place or at the wrong time. If, for instance, splashing be detected below the umbilicus at any time, there is either enlargement or prolapse of the organ, and if it be detected in any part of the abdomen after say a night's rest or seven hours after any meal, there is retention of contents pointing to deficient motor power or stenosis of the pylorus. The exact area, therefore, over which splashing can be detected should be noted, and inquiry should always be made as to the time which has elapsed since food and drink were last taken and as to their amount.

It is on palpation chiefly we depend for the detection of a tumour. If such be present, its size, shape, consistency, and position should be noted. It should be observed, also, whether the tumour be tender and the seat of pain, and whether it change its position with respiration and varying degrees of stomach distension. Stomach tumours move more or less downwards with inspiration, unless the organ be prolapsed, in which case they do not. The amount of movement depends largely on how much the patient uses his diaphragm in breathing. Tumours of

the pylorus move rather less on respiration than those of the body of the stomach, and the lower a pyloric tumour is situated the less it is likely to move. It is only necessary to recall how the positions of the stomach vary to realise that pyloric tumours show a like variation. Such a tumour may be felt beneath the right costal margin, or it may be lower down in the abdomen, though usually it is to the right of the middle line and above the level of the umbilicus. It is often difficult to determine whether a tumour be in connection with the stomach, and if so, with what part. This question may sometimes be cleared up by inflation or transillumination of the viscus—methods which will be referred to below.

Percussion.—It is only possible to partially outline the stomach by percussion, viz. those parts of it which are in relation to the thoracic and anterior abdominal walls. The empty stomach cannot be percussed at all. For percussion the viscus needs to contain some gas, and in its delimitation from neighbouring organs too much dependence must not be placed on a characteristic stomach resonance. The quality of this percussion note depends on the quantity of gas in proportion to the liquid within the organ and the tension of the contained gas. If the patient be not flat in bed, and the stomach be dilated, rather full, and contain but little gas, the lower portion of the organ will be dull, not resonant, on percussion. Unless this fact be remembered the organ will be mapped out as smaller than it is in reality. The determinations of the positions of the greater and lesser curvatures are the two most important points. The exact outline of the stomach resonance above the left costal margin is of less importance. In demarcating the lower edge of the left lobe of the liver from the stomach, it is necessary to percuss very lightly, or the lower edge of the liver will be placed too high.

Auscultatory percussion is of great use in delimiting the gastric area in the abdomen. When the stethoscope is placed over the stomach just on or immediately below the left costal margin, and the skin lightly tapped or stroked near the instrument, a characteristic note will be heard, due to the vibrations set up within the viscus. On tapping or stroking away from the stethoscope in different directions until this note changes, as it will when the boundaries of the stomach are passed, the outline of the organ will be defined.

Inflation.—This may be effected in two ways: by pumping in air, or by the evolution of carbonic acid gas within the viscus. In the first method the soft stomach tube is connected with an india-rubber bulb. With the second method the patient is made to drink a small teaspoonful of tartaric acid dissolved in four ounces of water, immediately followed by a large teaspoonful of carbonate of soda, also dissolved in four ounces of water. Each method has its advantages and disadvantages: the first enables the stomach to be distended as gradually

or slightly as is convenient, but it requires an apparatus, and is more likely to alarm the patient; the second is simpler in application, but it is not possible to regulate the degree of stomach distension.

Inflation of the stomach by gas is contra-indicated in cases of recent

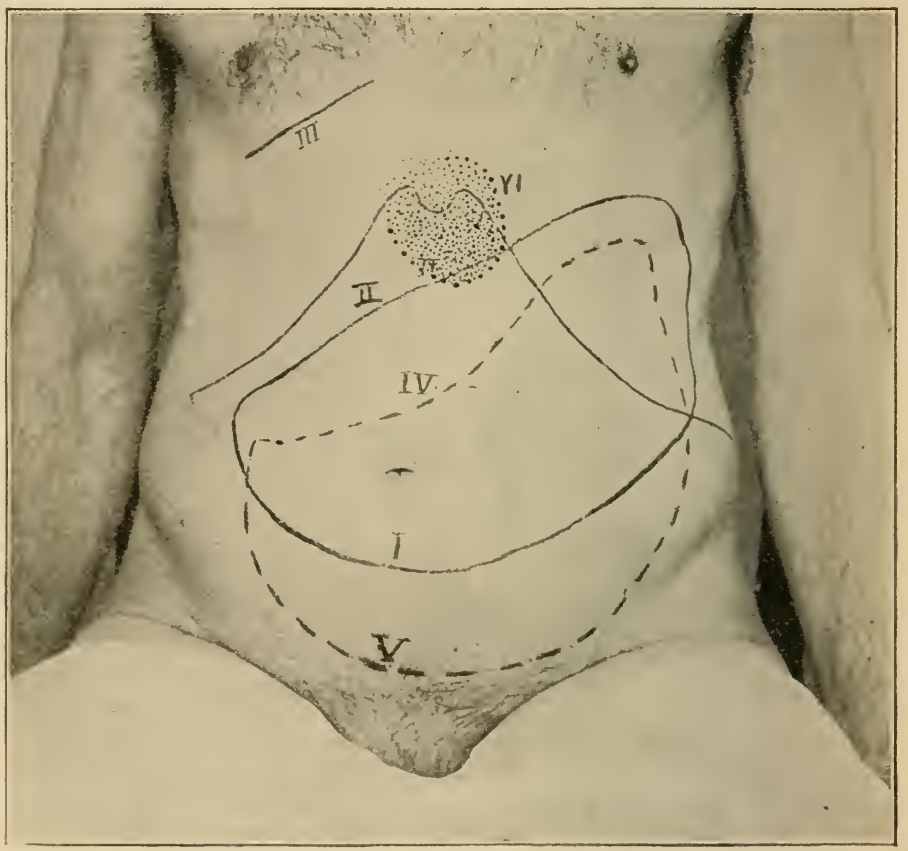

FIG. 10.—Diagram showing outlines of abnormalities in size and position of the stomach. I. Greater curvature of dilated stomach. II. Lower edge of liver, the dilated stomach passing beneath it. III. Upper boundary of liver dulness. IV. Lesser curvature of a stomach which is prolapsed (gastroptosis) as well as dilated (gastroectasis). V. Greater curvature of same stomach. VI. Area of superficial tenderness belonging to the 6th and 7th dorsal segments.

hæmorrhage, or where there has been recent manifestation of a gastric ulcer. Speaking generally the procedure is free from danger. If discomfort or embarrassment follow, two or three drops of cajuput or clove oil on a piece of loaf sugar will often alleviate, and if these fail the stomach tube can easily be passed.

Inflation of the stomach has many advantages. It enables the position and size of the stomach to be made out with certainty, it gives

some idea of the motor power of the wall, and whether there be pyloric obstruction or not. Further, it often enables an opinion to be formed as to the positions and relations of tumours. Thus a tumour of the right upper abdomen may after inflation be proved to be at the site of the pylorus ; or again, for instance, there may be a tumour in the upper part of the epigastrium, and inflation may show the stomach to be prolapsed and the tumour to be above the lesser curvature ; or, on the other hand, the tumour may still be within the stomach area after inflation, and then be proved to be situated in its wall.

It is always important to note the physical signs before inflation, so as to be able to observe the changes after. Tumours are sometimes more easily palpable after inflation, and at other times the reverse. Tumours of the pylorus usually move to the right and slightly down when the stomach is distended.

Gastrodiaphany.—This consists of lighting the interior of the stomach, and observing those portions of the abdominal wall which are transilluminated.

The apparatus employed for the purpose is a tube which resembles a stomach tube, except that the distal end is of glass (Fig. 11, 2, H). Within the glass end is a small electric lamp, which is connected with wires running along the interior of the tube, and ending in screw terminals. The latter are connected with a battery, a resistance and spring key being interpolated between them. Sometimes the apparatus is so arranged that a stream of water circulates round the incandescent lamp to keep the latter cool, but this is unnecessary, provided the lamp be only lighted for a few moments at a time by means of the spring key, and this is all that is needed.

The patient should be seated erect in a chair, and the room should be darkened. A previous experience with the ordinary stomach tube is desirable. It is best that the stomach should contain a small amount of liquid and gas. The right amount of resistance required to give a good light being first determined, the tube is introduced. The latter is then manipulated so as to change the positions of the distal end. Each time the position is changed the light is switched on, and the area of thoracic or abdominal wall illuminated is noted, and its boundaries marked with pencil.

This method is useful in determining abnormalities in the size and position of the stomach, and it sometimes helps in deciding whether tumours of the stomach region are in the wall of that viscus or not. It is, however, of limited utility. In many cases it is unnecessary, because all that it can tell has been learned from the usual and easier methods. Further, the method is not uniformly successful in application, and thus caution is needed in forming conclusions from it.

Röntgen Rays.—If the stomach be nearly or completely empty, and be inflated with air, the viscus may be seen as a light area through the

screen. The process of inflation may be watched and the outlines of the organ determined. The movements of the stomach have been studied by mixing food with large doses of bismuth, and observing the shadows produced by the latter. Although of restricted application X-rays on occasions might be of great assistance, *e.g.* in the detection of a tumour or subphrenic abscess.

Investigation of pain and tenderness.—Pain may be located in the stomach region, or be situated away from it, *e.g.* in the back. Careful inquiry should be made as to the position of pain, and of its relations to food and effort. Some pains (*e.g.* in ulcer) are started or accentuated by the ingestion of food, others (*e.g.* in neurasthenic dyspepsia) are relieved by it (see p. 201).

Tenderness is deep or superficial. Deep tenderness is brought out by pressure or percussion, and is proportional to the amount of force employed. It can only be elicited over that part of the stomach which is in relation to the anterior abdominal wall. It is important to note the intensity, the locality, and the area of such tenderness ; when the latter is well defined and limited to a small area (*e.g.* one or two finger tips), it is most frequently associated with a lesion of the interior wall of the stomach. Superficial tenderness is made manifest by very lightly picking up the skin between finger and thumb, or by lightly touching the skin with the blunt end of a pin. When a tender skin area is reached a greater sensitiveness is experienced, which may actually amount to pain ; over these tender areas the superficial reflexes are markedly increased. The areas of superficial tenderness associated with stomach lesions correspond to the sixth or seventh, eighth and ninth, and sometimes tenth dorsal segments (Head). Sometimes the tenderness will extend round the body from the middle line in front to the back, but most often it is limited to a small area on the anterior surface of the body, with sometimes in addition another such area on the posterior surface. Fig. 10 shows such an area, and in the case from which it was taken there was another small area on the back, a little below the angle of the left scapula. There is some reason to hope that the position of these areas of cutaneous tenderness may aid in the localisation of stomach lesions, and it has been held that tenderness in the sixth and seventh dorsal areas is suggestive of a lesion of the cardiac end, and that the ninth and tenth dorsal areas stand in relation with the pyloric end. These inferences cannot, however, be drawn with confidence. Cutaneous tenderness should be watched for, and, if present, mapped out, the degree of stomach distension and other observations being noted at the same time.

The foregoing methods have dealt mostly with anatomical abnormalities. It is now necessary to pass to those which elucidate perversion of functions.

Test meals.—The motor and secretory processes may best be in-

vestigated by the examination of the gastric contents at varying intervals after "test meals" of known composition.

A convenient test meal consists of finely-chopped beef or mutton, 6 ounces; stale bread or toast, 3 ounces; water, 10 ounces: or a lighter meal may be ordered, in which the meat is omitted, and 5 ounces of stale bread or toast given with half a pint of tea.

The lighter meal may be withdrawn from the stomach from one to one and a half hours after eating, and the heavier test meal three to four hours after. The latter is the more generally useful from the fact that it tests more completely the powers of digestion. A test meal should always be given in the early morning, as in this way it is more easy to ensure the stomach being empty. If there be any doubt as to this the organ should be washed out previously.

To form an opinion as to the motor function it may be necessary to withdraw the stomach contents at varying intervals after the meals, and sometimes to increase the quantity of the latter. After the larger test meal the stomach should be quite empty at the end of six hours, and at the end of four hours the contents should be small in amount, finely divided, and pultaceous. If otherwise, there is retention and the stomach is imperfectly voiding its contents, because either its motor power is insufficient or the pylorus is stenosed. If, on the other hand, the stomach be empty too soon, say two hours after the larger test meal, motility is excessive.

The stomach tube.—This consists of an india-rubber tube A, in the distal end of which are openings. These may be either situated laterally a short distance from the end, in which case the latter should be solid, or there may be a single large opening at the extremity of the tube. There are often in addition smaller oblique openings. The tube A is joined by means of a short glass connection with the tube B, and so to the funnel C. The tube A should be marked at 6 inches (*a*), $8\frac{1}{2}$ inches (*b*), and $16\frac{1}{2}$ inches (*c*) from the distal end, these points marking the cricoid cartilage, the left bronchus, and cardiac orifice as measured from the teeth. This is all the apparatus which is absolutely necessary. Fig. 11, 1, shows an elaboration in the form found useful by the writer; there the glass tube is also connected by a branch to the india-rubber bulb D, which may be employed to either suck air out or pump air in according to the end of the bulb, K or H, which is fixed to the glass tube. A clip serves to close the tube B when desired, and a tap shuts off the bulb from A. By this appliance it is possible to inflate the stomach with air or withdraw its contents at pleasure. If, as sometimes happens, the patient find difficulty in expressing the stomach contents, or the tube A get blocked, it is possible by means of D to aspirate and so overcome the difficulty.

The passing of the tube.—The patient should for choice sit erect in bed, or on a chair, and the head should be slightly flexed. He should

next be assured that there is no fear of the tube going the "wrong way" and be directed to go on breathing, and make an effort to swallow when

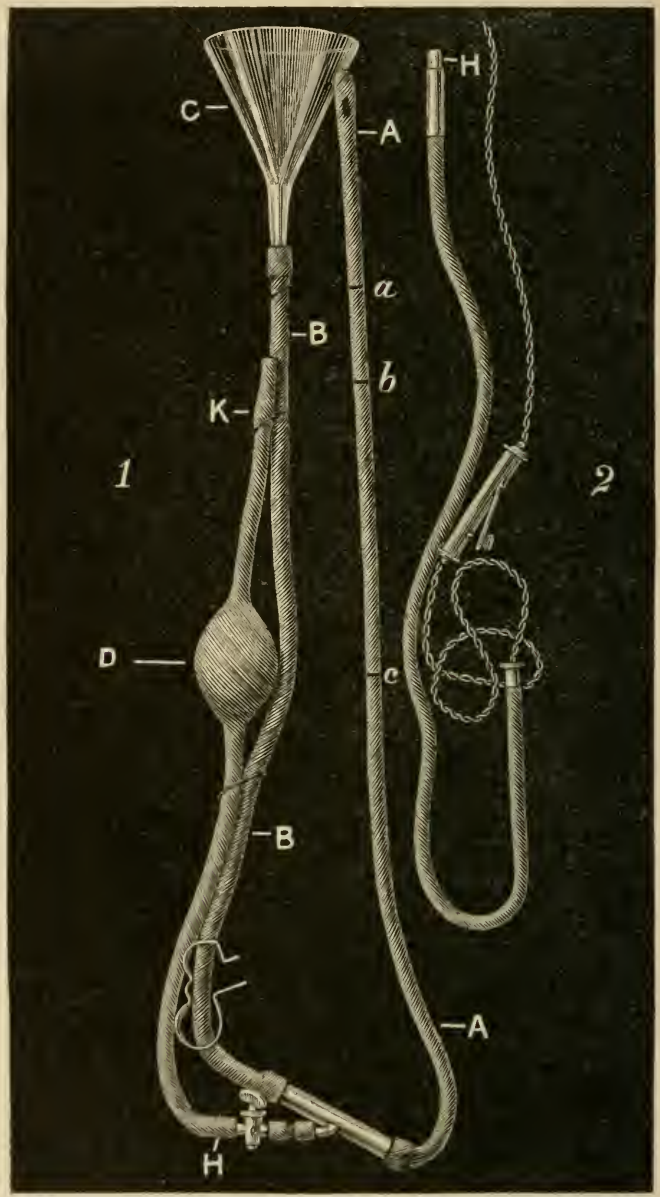

FIG. 11.—1. Stomach tube with attachment. 2. Gastrodiaphanoscope.

ordered to do so. The tube should be moistened with water, but not dipped in glycerine or oil, and passed down the pharynx, the patient being energetically commanded at this moment to swallow. By the

latter effort the tube is carried past the cricoid—the only likely obstacle. Now stop a moment and get the patient to breathe, and then pass the tube well into the stomach. It is very important that the breath should not be held. To obtain the contents get the patient to express the contents ("straining") by contracting the abdominal muscles. In some cases even this is not necessary, for when the tube reaches the stomach peristalsis commences. If the contents will not come or stop after having commenced to do so, the bulb D may be used either to draw the contents into the tube, or expel any large fragment which may be blocking the apertures in the end of it. Or if only a single tube and funnel are at hand sufficient water may be added to just fill the tube, the flow inwards being controlled by pinching the india-rubber; then the glass funnel should be quickly lowered and inverted, and the syphon action will be started. When withdrawing a test meal it is not necessary to completely empty a stomach, but it is very desirable to accomplish it without the addition of water.

The passing of the stomach tube must in certain cases be avoided, or undertaken with caution. After severe gastric hæmorrhage, or recent manifestations of ulcer, and in certain cases of aneurysm, it should be avoided. Pause should be made also when the heart is diseased, or the patient very weak and exhausted.

Physical examination of the intestines.—Inspection will show if the abdomen be distended, and, if so, whether uniformly or especially in certain regions. Thus a distended large intestine would show itself on each side of the abdomen and across the upper half, whereas distended coils of small intestine would arrange themselves more in the centre of the abdomen and in horizontal positions, one below the other. The mobility of the abdominal wall should be noted; loss of mobility is a valuable sign in peritonitis and some other diseases.

Distension of the cæcum calls for special remark on account of the frequency of its occurrence, and the number of conditions which give rise to it. It manifests itself, if the patient be spare, by a visible swelling, which is elastic on palpation and resonant on percussion if full of gas, and doughy and dull if full of fæces.

While dealing with this part of the abdomen it is well to note that in some cases the appendix is palpable. It may at the same time be found to be tender—a fact which might suggest a catarrh of the appendix, and possibly forbode an acute inflammatory attack. Under these circumstances careful inquiry as to previous attacks of pain should be made, and the existence of superficial tenderness investigated (see p. 349).

It is important to remember that fæces may become impacted and be mistaken for tumours. They may be detected by their doughy and inelastic feel, by the change in their shape, which can sometimes be brought about by steady pressure, and by their changes of position. In all cases of doubt a large enema should be given.

Examination of the rectum.—The patient should lie on the left side with the thighs and legs fully flexed, the head should be near the left side of the couch, and the buttocks near the right. The observer should see that the nail of his right forefinger is short; he should fill the nail with soap and vaseline his finger; the anus should have a little of the same lubricant applied, and the finger should be passed into the rectum, the patient being directed to bear down the while. The examination should be systematic, first the posterior wall, then the lateral walls, and finally the anterior. It should be noted if the rectum have tone, or whether it be lax and dilated (so-called ballooning). In the male the prostate will be felt projecting through the anterior wall, and higher up the vesiculæ seminales. In the female the cervix uteri will be felt, and in cases of retroversion the body of the uterus. This latter may be easily mistaken by a beginner for a morbid growth. Folds of mucous membrane are often met with, and may give rise to erroneous impressions, unless it be remembered that their presence is not abnormal.

Owing to the frequency with which malignant growths are situated in the rectum, digital examination is a matter of importance. These tumours are, however, sometimes situated beyond reach of the finger, so that inability to feel a growth would not justify the statement that such did not exist.

EXAMINATION OF STOMACH CONTENTS

These may be vomited or withdrawn by the tube, and are investigated macroscopically, microscopically, and chemically.

Naked-eye appearances.—First, note should be taken of the quantity, and of its relation to the amount of food taken at the last meal, and the length of time which has elapsed since the meal. Seven hours at latest after a meal the stomach should be empty, and the contents should be materially diminishing two hours before this. If such be not the case—if, for example, any part of a supper eaten at night can be vomited or withdrawn in the morning—then there is retention of contents; it may be from impaired motor power or pyloric obstruction.

Again, if the quantity of food returned from the stomach exceed that taken at the preceding meal, there must be undue retention of contents. This would not necessarily hold if the fluid only were moderately increased, for it has been shown that the gastric mucous membrane can, under certain conditions, secrete water into the viscus. The occurrence in the vomit of some article of diet (*e.g.* peas) which the patient partook of several meals previously is evidence in the same direction. The next thing to notice is the consistency of the contents. It may be there are coarse fragments of half-digested meat. The significance of these depends on the lapse of time since their ingestion; if the interval be four hours or more it means that peptic digestion is deficient. On the

other hand, the contents may be uniformly grumous and devoid of any solid particles, or they may be admixed with an excess of tenacious mucus, pointing to gastric catarrh, or be frothy, pointing to fermentation and stagnation.

A convenient way of observing the naked-eye appearances of stomach contents is to pour them into a cylindrical or conical glass vessel. Sometimes they distribute themselves into layers ; if there have been stagnation and consequent fermentation the top layer will be frothy from the gas bubbles contained in it. The smell of gastric contents may afford guidance. In digestion the contents should possess but little smell ; on the other hand, the sour acid smell of the vomit in cases of dilated stomach is very characteristic. The odour and significance of fæcal vomiting are too well known to need more than mention.

Blood is seldom present in stomach contents withdrawn by the tube—it is almost always vomited. It may appear as pure blood, and if so, the latter is usually dark and clotted. At other times the blood is more intimately mixed with the food, giving the appearance of coffee-grounds, due to the conversion of the hæmoglobin into hæmatin by the gastric juice.

Microscopic examination.—This is of less importance than the foregoing. A drop of unfiltered contents is mounted on a slide either alone or with a drop of very dilute iodine ; or a small portion of residue is taken from the filter and mounted in saline solution ; or again, a drop is dried and fixed and stained with *very* dilute gentian violet or methylene blue. After leaving the stain in contact with the film for three minutes, wash in water, dry, and mount in Canada balsam.

The preparations show particles of food, muscle fibres, starch granules, and fat globules, but the microscope tells us nothing more about them than can be learnt by the naked-eye examination.

Many micro-organisms may be seen. Of these the following deserve mention :—

(a) *Torulæ.*—Seen as cells, either singly or in chains. If the yeast cells be present in large numbers, and budding actively, it is evidence that the stomach contents have been stagnant. Yeast stains well with methylene blue.

(b) *Sarcinæ.*—These are micrococci grouped together into packets of cells resembling tiny bales of wool. They are numerous in cases of non-malignant dilatation, as distinct from gastroectasis caused by cancer, in which they are seldom found. Apparently carcinoma creates a soil unfavourable to their growth, for Oppler introduced pure cultures of sarcinæ into a carcinomatous stomach, and they had all disappeared in twenty-four hours.

(c) *Oppler-Boas bacilli.*—These are large bacilli, and may be seen easily in both unstained and stained preparations. They have the power of forming lactic acid from sugars, and their presence has the

same significance as that of lactic acid. They are found in large numbers in the contents of cancerous stomachs, and though their abundance is not diagnostic of malignant disease, it affords evidence in that direction.

Very rarely fragments—cell nests—of carcinomata have been discovered in a preparation, a valuable help to diagnosis, though not often forthcoming.

Analysis of stomach contents.—Analysis of the stomach contents informs us as to the composition of the gastric secretions and the products of their activity. For practical purposes it limits itself to determining whether, on the one hand, the essential ingredients, free hydrochloric acid and pepsin, are present and in proper quantities; and, on the other hand, to the search for such fermentation products as lactic, butyric, and acetic acids. The most important fact to ascertain is whether free hydrochloric acid is present at the height of digestion, and, if so, in what quantities. After the ingestion of food the hydrochloric acid secreted by the gastric glands partly enters into combination with the albumens of the food, but experiments have shown that when peptic digestion is normal, there is, above and beyond what is required to satisfy the affinities of these albumens, an excess of hydrochloric acid which is free—that there is, in short, enough hydrochloric acid and to spare. The presence of this free acid thus becomes a very convenient criterion of efficient peptic digestion, it speaks not only for itself, but for the efficiency of the gastric secretion as a whole. Where it is much diminished or absent peptic digestion is impaired. In the event of the free acid being absent a further measure of the digestive inefficiency might be obtained by determining the amount of hydrochloric acid in albuminous combination. This procedure, however, would lengthen the chemical investigation—a fact which would often stand in the way of its clinical application. Absence or marked diminution of free hydrochloric acid is seen in carcinoma, atrophy of the gastric glands, acute gastritis, and some cases of neurasthenic dyspepsia; and, on the other hand, hyper-secretion of free hydrochloric acid is associated with definite symptoms, and is a feature of many cases of gastric ulcer.

A separate search for pepsin is seldom necessary, since the presence of free hydrochloric acid is an index of its presence. Some pepsin may, however, remain when the free acid has entirely disappeared. Complete absence of pepsin would denote a graver derangement of function than the absence of free hydrochloric acid alone.

Organic acids, such as lactic, butyric, and acetic, are not present in appreciable quantities during normal digestion, and their presence may be taken as evidence of fermentation. Lactic acid, which is the most important of these acids, plays no essential part in the changes in the stomach. It is either introduced with the food in the form of sarcolactic acid, or its presence is the result of fermentation set up by micro-organisms. Active pepsin and hydrochloric acid secretion checks lactic

acid formation, and absence of these favours it. The marked presence of lactic acid is evidence of retention and stagnation of the stomach contents. This may be the result of failure of motor power, or of pyloric stenosis. Thus lactic acid is often found in cases of cancer, because the latter is so frequently the cause of pyloric stenosis, not for any other reason. The presence of butyric and acetic acids has the same significance as that of lactic acid.

Methods.—The first step is to ascertain by means of litmus paper if the contents be acid. This being so, we need to know to what the acidity is due, whether to (*a*) free hydrochloric acid, to (*b*) hydrochloric acid in combination with albumens, to (*c*) organic acids (lactic, butyric, and acetic), or to (*d*) acid phosphates. Of these factors *a* and *c* are volatile at the temperature of the water bath, whereas the remaining two are not.

Tests for free acids (hydrochloric, lactic, butyric, etc.).—(1) Dip a piece of congo-red paper into the stomach contents—a blue colour denotes the presence of free acid. This paper is as convenient as litmus paper, and may be obtained like the latter in booklets, or it may be made by saturating filter paper with a watery solution (1 in 1000) and allowing it to dry. Congo paper is an excellent test. It is quickly applied, and equally well to unfiltered as to filtered contents. It does not, however, differentiate with certainty between free hydrochloric acid and organic acids. The latter are, however, seldom present in large enough quantities to give the test in the same definite way which hydrochloric acid does.

(2) Take 5 c.c. of filtered contents in a test tube and add a pinch of calcium carbonate. Occurrence of effervescence denotes the presence of free acid (Hutchison).

Tests for free hydrochloric acid.—Mix a few drops of filtered contents with an equal quantity of Günzburg's reagent [1] in a small porcelain dish. Evaporate on a water bath, or, if quickness be necessary, with caution over the flame, removing the dish from time to time and blowing over the contents so as to avoid too great heat and consequent charring. When evaporation is completing a pink colour will appear round the margin of the residue if free hydrochloric acid be present and spread over the whole surface.

(3) Töpfer has introduced a simple test. It consists of a ·5 per cent alcoholic solution of dimethyl-amido-azo-benzol. A drop of filtered contents is placed on a white porcelain surface, and near it a drop of the above solution. The two drops are brought in contact. When free hydrochloric acid is present a cherry-red colour appears at the line of

[1] This reagent is best made by dissolving 2 grammes of phloroglucin, 1 gramme of vanillin in 100 c.c. of 80 per cent alcohol. It should be kept in a dark coloured bottle, and be recently prepared, on account of the readiness with which it decomposes.

junction. Although lactic acid will also yield this test, it will only do so, it is said, in a concentration above that which ever exists in the stomach.

[1] *Qualitative and quantitative determination of total acidity, free hydrochloric and organic acids.*—The filtrate is stirred with a glass rod to ensure uniformity, and two equal volumes, 10 c.c. each, are drawn off and placed in similar, wide porcelain basins. One portion (A) is evaporated to dryness on the water bath to drive off the volatile free acids; the other portion (B) is not evaporated. The residue from A should remain on the water bath for an hour or more and then be dissolved in 10 c.c. of distilled water. A drop should be tested for free hydrochloric acid, and if the latter be present the evaporation should be repeated.

To the contents of the two basins are now added four drops of a neutral litmus solution. They are then titrated with a solution of caustic soda, of such strength that 1 c.c. will neutralise 1 milligramme of hydrochloric acid. The object of the titration being mainly comparative, great care should be taken that the final tint is the same in both basins. Suppose that this is attained when A has received a c.c., and B has received b c.c. Then if $b = a$ there is no free acid that is volatile on the water bath; if they are not equal, then $b - a$ is the measure of the free acid originally present in B or A, which has been expelled on the water bath, and the amount of this free acid, expressed as hydrochloric acid, is $(b - a)$ milligrammes per 10 c.c. of filtered fluid.

We now proceed to compare the quantities of chlorine in the two neutralised solutions. For this purpose four drops of a freshly-prepared solution of potassium chromate must be added to each basin, and the contents carefully titrated with a corresponding solution of silver nitrate, 1 c.c. of which will exactly precipitate the chlorine in 1 milligramme of hydrochloric acid. As before, the object in view is mainly comparative, and this is secured if the final point, which is known by the appearance of the brick-red colour of silver chromate, be the same in both basins. Suppose that A has taken α c.c. and B has taken β c.c. before this end point is reached. Then $\beta - \alpha$ is the measure of the free hydrochloric acid which has been expelled on the water bath, and amounts to $(\beta - \alpha)$ milligrammes per 10 c.c. of filtered fluid.

It may be pointed out that one titration is to some extent a check on the other, for if free hydrochloric acid be the only volatile free acid present, then $b - a$ should be equal to $\beta - \alpha$. If they are not equal, then $(b - a) - (\beta - \alpha)$ will be the measure of the free volatile acids other than hydrochloric which are present, *e.g.* butyric acid. If expressed, for convenience, in equivalents of hydrochloric acid, this measure will be $\{(b - a) - (\beta - \alpha)\}$ milligrammes per 10 c.c. of filtered fluid.

[1] For this and the following section the writer is indebted to Mr. Hugh Candy, B.A., B.Sc., Lecturer on Organic Chemistry, and Analyst to the London Hospital.

The number of milligrammes per 10 c.c. is also the number of grammes per 10,000 c.c., and when divided by 100 will give the number of grammes per 100 c.c. of filtered fluid.

We have, therefore, the following data :—

Total acidity $= \frac{b}{100}$	grammes of HCl per	100 c.c. of filtered fluid.
Non-volatile acidity $= \frac{a}{100}$	,,	,,
Therefore volatile acidity $= \frac{(b-a)}{100}$	,,	,,
But free HCl $= \frac{(\beta-a)}{100}$	,,	,,
$\therefore$ volatile acids other than HCl $= \frac{(b-a)-(\beta-a)}{100}$	,,	,,

The non-volatile acidity, *a*, will include acidity due to acid salts, and to hydrochloric acid in albuminous combination. It is possible to estimate the latter, but the lengthening of the process thus involved is not sufficiently balanced by practical advantage.

The volumetric solutions employed may be conveniently prepared from the ordinary decinormal solutions by diluting 100 c.c. of the latter with distilled water till the bulk measures 364 c.c.[1] Their use is recommended both because it reduces the margin of error and because it dispenses with arithmetical labour.

Determination of organic acids.—The total quantity of these is determined by the foregoing method. Their presence is shown by extracting them with ether. Take 10 c.c. of filtered contents and shake well with 20 c.c. of ether in a separation funnel. Allow the liquids to separate, draw off the lower stratum of filtered contents and again shake with 20 c.c. of ether. Again draw off the contents and repeat the process with a third quantum of ether. Take the 60 c.c. of ethereal extract and equally divide it between two beakers. Place one of these on a water bath and evaporate the ethereal contents to dryness. At the temperature of the water bath the butyric and acetic acids will evaporate, but the lactic, if present, will be found in the residue. Dissolve the residue in water and add a few drops of it to Uffelmann's reagent, when the blue colour of the latter will be changed to yellow if lactic be present. Uffelmann's reagent is made by mixing 20 c.c. of distilled water with 10 c.c. of a (1 in 20) solution of carbolic acid and adding one drop of liquor ferri perchloridi. The solution is of an amethyst blue colour, and should be made fresh each time.

Allow the contents of the second beaker to evaporate at the temperature of the air. Butyric acid may be recognised by the characteristically

[1] 36.4 is the molecular weight of hydrochloric acid.

rancid smell of the residue, or if the latter be taken up with water and a small fragment of chloride of calcium be added, the acid will separate as a drop of oil. Acetic acid may be detected by neutralising the aqueous solution of the residue with carbonate of soda, and adding one or two drops of weak perchloride of iron solution, when the claret-coloured acetate of iron will appear.

Presence of pepsin.—It is only necessary to test for this when free hydrochloric acid is absent.

The quickest way is to search for albumoses by neutralising 5 c.c. of the filtered contents and applying the biuret test. If these are present pepsin must be.

Or the presence of pepsin may be shown by the digestive action of the filtered contents on egg albumen. Stamp out some very small and thin discs of white of egg of equal size. Place one of these with 5 c.c. of filtered contents in each of three test tubes *a*, *b*, and *c*.

To *a* add nothing further, to *b* add two drops of dilute hydrochloric acid, and to *c* add two drops of acid and two grains of pepsin. Place the three tubes in an incubator for three hours. If there be no solution of the disc in *a* or *b* pepsin is absent.

BERTRAND DAWSON.

Special Symptomatology and Treatment of Gastro-Intestinal Diseases[1]

INFLAMMATION OF THE STOMACH AND BOWELS

Acute Gastritis

Symptoms.—Having regard to the wide range of cause, from an indiscretion in diet, to a single dose of an irritant poison, or the invasion of pyogenic micro-organisms, it is to be expected that a considerable variation in the intensity of symptoms is exhibited by the cases comprised under this term (see pp. 156-163).

In the milder cases, when the condition supervenes upon previous good hea th and the cause is a temporary one, the patient may complain of but a slight feeling of malaise with disinclination for exertion, and an elevation of temperature by a degree or a degree and a half, or perhaps not even so much. The symptoms otherwise are chiefly referable to the stomach, as loss of appetite and thirst; a sense of discomfort even amounting to pain in the epigastric or left hypochondriac regions; a feeling of nausea, and probably retching or vomiting at first of such partially digested food as may be in the stomach, and later of mucus and sour fluid, examination of which shows an absence of hydrochloric and the presence of organic acids, lactic, acetic, or butyric. The tongue is coated and foul, and the breath offensive. The bowels may be unaffected, or there may be some diarrhœa. Of symptoms remote from the digestive organs, a slight degree of headache, frontal or occipital, and a somewhat quickened pulse are the most usual.

According to the character and intensity of the exciting cause, any or all of these symptoms may be much increased in degree. The slight sense of illness may deepen into a state of prostration, and such is specially likely to be the case in children, in whom also the intestine is somewhat more prone to be simultaneously affected. The temperature may reach 103° or even higher, this being in some

[1] For permission to make considerable extracts from his articles on Diseases of the Stomach and on Diseases of the Intestines, which appeared in the *Encyclopædia Medica*, vol. xi., and in Quain's *Dictionary of Medicine*, 3rd edition, the writer is much indebted to the courtesy of Messrs. W. Green and Sons, and of Messrs. Longmans, Green, and Co., the respective publishers of these two works.

measure determined by the temperament of the patient. The pain may be severe, of a burning character, and shooting through to the back, increased by deep breathing or by anything but the blandest material swallowed. Tenderness also may be elicited over the epigastrium, and the abdominal muscles are rigid or contract on the slightest pressure.

When the gastritis is the result of a corrosive poison, pain is severe, and is felt in the throat and gullet as well; the retching and vomiting are most distressing, and the material brought up is frequently blood-stained with shreds of the mucous membrane; the motions may also contain altered blood, and defæcation may be painful. The general symptoms are much intensified, the patient becomes cold and collapsed, covered with a cold sweat, with a small, scarcely perceptible pulse and rapid shallow breathing, increasing until death occurs from cardiac failure, sometimes with convulsions or coma; or there may be peritonitis, to which the fatal result is attributable.

In *phlegmonous or suppurative gastritis* the symptoms are also severe, especially those indicative of the general condition, but none are peculiar to the disease. There is a state of grave illness which sets in suddenly and rapidly deepens, and death commonly ensues in the course of a few days, though occasionally the disease has been known to continue for weeks. The temperature ranges higher as a rule than in other forms of gastritis, and may reach and remain at 104° Fahr. or even higher; the nervous symptoms, such as severe epigastric pain, restlessness, delirium, and coma, are considerable; jaundice may develop, and vomiting, constipation or diarrhœa, a dry brown tongue, thirst, and loss of appetite are constant. When the suppuration is circumscribed and an abscess is formed in the gastric walls, pus may appear in the vomit from a bursting of the same, but short of the occurrence of this phenomenon, the diagnosis will remain uncertain, though the nature of the case may be suspected.

Recovery, except in the severest forms, is usually rapid and complete, although, should the cause be frequently repeated or persistent, the condition passes into a subacute or chronic state.

Chronic Gastritis

The symptoms of *chronic gastritis* are essentially those of a chronic gastric dyspepsia, since that is the usual morbid state

underlying the clinical complex known as chronic gastric indigestion, when that malady is not purely nervous in origin. It may become established as a sequence of acute gastritis, or may slowly develop without any definite commencement as the result of obstructed portal circulation, of continued infection from oral sepsis, or in the course of chronic toxæmias, such as gout, chronic nephritis, etc.; but its most frequent cause is probably to be found in the continued maldigestion of food in the stomach, whether from imperfect secretion of gastric juice, impaired motility of the organ, or oftenest of all from the repeated subjection of the mucosa to improper ingesta. Hence, besides occurring alone, it is an almost constant accompaniment of a dilated stomach, and commonly is found with other gastric diseases, such as ulcer and cancer. The general result of the structural changes which characterise a chronic catarrh of the gastric mucosa is to impair the movements of the organ, to lead to a secretion of juice which is deficient in quantity or in hydrochloric acid, and probably to set up an undue sensitiveness of the membrane. Once established, therefore, chronic gastritis tends to produce or to intensify those conditions to which its occurrence may have been due. As might be supposed from the varied circumstances attending its causation, the symptoms it gives rise to are varied in range and character, from the mildest degree of discomfort to almost continuous suffering. Broadly speaking, the condition is one of delayed digestion, marked by a sensation of weight or oppression, or even slight pain that comes on some time after food, and may continue for several hours. There may be some tenderness, especially on pressure over the stomach, and the exertion even of deep breathing may cause discomfort in that region. The appetite is indifferent or variable and capricious. Flatulence with eructations of gas that are frequently induced with difficulty, though affording considerable relief when they have occurred, are among the most constant indications. There is a general liability to nausea, especially in the morning, and vomiting also is apt to be most frequent then, due, as it often is, to the absorption of poisonous products of digestion. Indeed, auto-intoxication is most probably the explanation of many of the remote symptoms of this condition, such as the dull, heavy headache so constantly met with, the lassitude and disinclination for mental and bodily exertion, muscular cramps, and vertigo. The vomit or material artificially withdrawn from the stomach shows a deficiency of hydrochloric acid and excess of mucus; the results of proteid digestion may be recognised, and also the products of carbohydrate fermentation. The appearances of the

tongue are less indicative of the state of the stomach and digestion therein than was formerly supposed; it may be thickly coated, or large, flabby and indented with the teeth and clean, or red and denuded; such variations are more dependent on the state of the mouth, teeth, and mode of breathing, and all may be met with in the course of a chronic gastric catarrh. A nasty taste in the mouth and foul breath are usually complained of. The ill-effects of the indigestion on the general nutrition are seen in the wasting, the muddy complexion, the anæmia, and the condition of the urine, which is frequently scanty, high-coloured, and concentrated, throwing down lithates, or uric acid, or oxalate of lime crystals. The digestive processes in the intestine are more or less upset by the ill-prepared food in the stomach, and constipation, with attacks of diarrhœa, flatulence, and other symptoms, complicate the initial gastric disorder. All degrees and combinations of the conditions enumerated characterise the various cases of this very common complaint.

In patients suffering from this disease the examination of the stomach-contents is often of very valuable assistance. In making the diagnosis, attention should be paid to the possible existence of other stomach affections, or of general diseases, such as gout, etc., and still more to the habits and diet of the patient. Upon the nature of these several circumstances and the steady pursuance of treatment the prognosis of the case will mainly rest.

Treatment.—The ordinary case of *acute or subacute gastric catarrh* requires no treatment as a rule, beyond entire abstinence from food for twelve or twenty-four hours, washing out the stomach by drinking warm water freely, and a mild mercurial purge. If there be much pain, an effervescing mixture containing liquor bismuthi et ammoniæ citratis, with hydrocyanic acid, will be required, and if it be clear that the stomach is empty, and yet retching and vomiting be excessive, a quarter of a grain of cocaine with one drop of carbolic acid in a mucilaginous draught will be found very useful. A hot bottle or fomentations to the abdomen will give great comfort.

In the more serious forms of gastritis following irritant poisons, appropriate antidotes will be required, and probably opium for the relief of pain. The general state of the patient demands attention, and stimulants or strychnine to avert fatal collapse. Except in this same direction, little can be done for the very serious phlegmonous form of inflammation.

Such food as is required should be of the simplest character;

milk and soda water, thinly prepared plasmon or Benger's food, are quite sufficient. A cup of tea is one of the earliest things asked for in the recovering cases, and as a rule may be allowed.

To ascertain and if possible remove the cause is the first and most important step in the treatment of a *chronic gastritis*. When this is due to errors in diet, whether of quality or quantity, alcoholic excess, insufficient mastication, or the state of the teeth and gums, correction of the fault may of itself be soon attended with cure. So satisfactory a result cannot be looked for when the catarrh is the consequence of a chronic congestion from portal obstruction, or is the accompaniment of dilatation or cancer of the stomach, and palliative measures alone are possible.

The indications for treatment are threefold—to do what is possible to restore the damaged mucosa, to make up for the deficiencies in the digestive process, and to treat the symptoms to which these conditions give rise. Working as they do in a vicious circle, relief at any point contributes to the general improvement. If complete rest could be given to the stomach, the chance of its self-restoration to a normal state would doubtless be much advanced, but this is of course impossible, as the patient must be fed, and in such cases the demand for nourishment is often especially great. General tonics, change of air, and treatment at chalybeate or other spas, *e.g.* Harrogate, Homburg, Ems, Vichy, etc., where the dietetic requirements are enforced, are called for in severe or long-standing cases. Bismuth and nitrate of silver are the drugs that probably exert the most directly beneficial effect on the mucosa, and it is said that their efficacy is increased by their intragastric application in the form of spray. Carlsbad salts are a useful adjunct to any treatment that is adopted. Digestive deficiencies may be in great degree remedied by regulation of the diet, and by the administration of hydrochloric acid and pepsin with vegetable bitters, since in chronic catarrh the active agents of the gastric juice are commonly diminished in quantity.

In all cases the habits of the patient should be ascertained and the regimen adopted accordingly; the individual as well as the disease must be considered in the treatment laid down.

Acute Enteritis

Within this term are included all those cases where the essential features of an inflammation of the small intestine are present, vary-

ing in severity from a simple catarrh or muco-enteritis to those graver forms possessed of special features, such as suppuration and ulceration. The more severe cases, especially in children, are sometimes called simple or English cholera—cholera infantum. The distribution throughout the bowels of the several forms of inflammation and the morbid changes by which they are characterised have been described on pp. 155 to 163.

Symptoms.—That a considerable variation is met with in the kind and severity of the symptoms presented in cases of enteritis is only to be expected when the great difference in degree and extent of morbid change that is met with is remembered, as well as the great variety of causes; and in a very large number of cases the manifestations are quite out of proportion to the appearances found *post-mortem*. Whereas one patient may suffer from an attack of intestinal catarrh, with but a trifling array of symptoms, another may succumb within a few days. There is no one symptom or even group of symptoms that is absolutely characteristic of the disease; even the general state of the patient is not constant, and the actual existence of the condition is often assumed, without anything like proof.

The temperature may reach 102° Fahr., or even higher; or in some cases it may be scarcely elevated. The appetite may be unaffected, especially if the upper part of the tract be free from the disease; while there may be complete anorexia when the reverse is the case. Thirst is of usual occurrence, and it becomes very marked when the evacuations are abundant and fluid. The tongue indicates rather the general state of the patient, and is even a less reliable index of the actual state of the intestinal mucous membrane than in corresponding affections of the stomach. It may be dry, red, irritable, and glazed; or coated with a thick fur, with the edges and papillæ bright and prominent; in milder cases it is often unaffected. The urine may contain a large amount of indican, indicative of albuminous putrefaction in the intestines. The character of the pulse varies with the general state. If the pyrexia be extreme, there is the usual dry skin and concentrated urine, with a tendency towards the production of the "typhoid state," which is usually reached in fatal cases. In many cases the prostration is excessive, though the mind is generally unaffected to the end, and very often there is a marked irritability of temper. Headache is of rare occurrence in enteritis. A persistent hiccough is met with sometimes. In children the disease rapidly leads to a condition of emaciation and collapse. The child lies in a languid, almost

torpid, state, with the skin of the abdomen intensely hot and dry; while the extremities are cold and blue, the face is pinched, and the body generally appears shrunken. Frequently the state is interrupted by attacks of convulsions, especially if dentition be in progress; or the child is extremely fretful, and maintains an almost constant, short, feeble cry, evidently accompanied by pain.

In cases of so-called "ptomaine-poisoning" the symptoms supervene within a few hours after the meal, and run an extremely severe course, frequently terminating in death, although a great variety is to be noticed among different individuals exposed to the same cause. In addition to the evidences of a virulent gastro-enteritis, such as vomiting, diarrhœa, bloody stools, abdominal cramps, pyrexia, and later, prostration and collapse, certain nervous phenomena—convulsions, tetanic spasms, paralysis, and coma—are frequently present in varying degree.

Stools.—Diarrhœa is in some respects the most constant symptom, though, when the affection is limited to the higher part of the canal, and space is given for the re-absorption of the excessive exudation, it may not only be wanting, but there may be actual constipation. The lower down the bowel is inflamed, the greater the liability to diarrhœa. In the severe forms of the affection complete constipation may be due to the paralysis of the inflamed bowel, arresting the peristalsis, and allowing of the accumulation of the intestinal contents above the lesion. This is obviously more complete when the enteritis is associated with any state producing mechanical obstruction, such as intussusception.

The character of the evacuations is very variable. As a rule they are semi-fluid when diarrhœa exists, or they may chiefly consist of an almost clear liquid with a few fæculent flakes; but, when time has permitted a partial re-absorption of the fluid portion, the stools become more consistent. In catarrhal affections of the small intestine, mucus occurs in but small amount, and is seldom to be recognised by the naked eye. Sometimes bile-stained particles may be detected. Blood is not usual except in the severer forms, unless there be ulceration or hæmorrhoids; and pus is seldom noticed. Quantities of intestinal epithelium, crystals of triple phosphates, and micro-organisms, especially the bacillus coli, may be detected by the microscope.

Owing to the imperfect performance of digestion or absorption, the motions are liable to contain many abnormal constituents—as fat, when the duodenum and upper part of the jejunum are involved, or even masses of food scarcely changed; and the altered characters

of the intestinal contents, with the products of decomposition, are in themselves most effective in maintaining a diarrhœa. As a rule, the discharges are paler than normal, or may be even colourless. The greenish tint so often seen in the enteritis of children is due either to a pigment formed by a bacillus, or to biliverdin. In the latter case it is considered as indicating some abnormal alkaline change in the intestine, whereby the biliverdin has become altered, though the stool itself as passed is acid in reaction. Unaltered bile-pigment in the fæces is abnormal, and its presence indicates catarrh high up in the small intestine, being generally associated with fluid evacuations from increased peristalsis. The odour is usually extremely offensive or even putrid, especially the white or grayish putty-like masses passed by children, though sometimes, when the motions are very liquid and colourless, smell may be altogether absent.

Owing to a large production of gases, discharges of flatus are of very frequent occurrence, but unless there be actual obstruction, tympanitic distension of the abdomen is not usual.

Vomiting, except in the severe forms, is not a common symptom of enteritis, unless the stomach be involved, and it is relatively more frequent in children than in adults. Short of actual vomiting, nausea is frequently complained of.

Pain and tenderness.—Pain in itself is a most uncertain symptom, being scarcely noticeable in the milder forms of catarrh. When the peritoneum is involved, the pain and tenderness are marked and characteristic. Both may be generally diffused over the abdomen, or may be local in character. In a large number of cases the pain is referred to the umbilical region.

Diagnosis.—The variability and oftentimes vagueness of the symptoms frequently admit of a diagnosis of enteritis being made only by a process of exclusion. The history of improper feeding, whether temporary or prolonged, often suggests the nature of the disease, though it cannot be denied that all rules of a rational dietary are frequently violated with apparent impunity both by children and adults.

Diarrhœa alone can by no means be taken necessarily to indicate the existence of intestinal inflammation, and the same may be said of constipation, pain, vomiting, and other single symptoms. It is rather to a group of symptoms, with the previous history, that the observer must look. The character of the stools, as already described, often indicates the region of gut affected; and the existence of extreme tenderness and pain, with a hard quick pulse,

and the characteristic decubitus, point to the involvement of the peritoneum, which an exacerbation of temperature tends to confirm. Inflammation of the duodenum, apart from a similar condition of the stomach or of the intestines, is of doubtful occurrence clinically, and gives rise to no symptoms by which it can be certainly diagnosed. Simple catarrhal jaundice is commonly attributed to a mild duodenitis; but there is no reason to regard the duodenum as the sole part of the canal affected, and of the supposed occurrence of inflammation in this situation as the result of toxic excretion in the bile very little is known. To distinguish between inflammation of the jejunum and ileum is usually impossible, nor, practically, is it a matter of importance. The history of the case, the course of the temperature, and the characteristic rash and headache, as well as Widal's reaction, should serve to separate typhoid fever from acute enteritis, which is sometimes mistaken for it.

Prognosis.—A simple intestinal catarrh occurring in a healthy subject certainly tends, after a few days, to complete recovery; but occurring, as it frequently does, in persons in ill-health, it is far more liable to pass into an obstinate chronic condition. In children the opinion should be very guarded. For while in a large number of cases perfect recovery follows removal of cause and suitable treatment, others, for no very apparent reason, will, in spite of everything, progress to a fatal termination; and this is, of course, more likely to be the result where the child is weakly or the subject of other disease. The mildest diarrhœa in children suffering from athrepsia quickly becomes serious, killing as it were from rapidly supervening shock. The onset of so-called summer diarrhœa during convalescence from such diseases as whooping-cough or pneumonia is also of grave import; and in true cholera infantum, great prostration, and especially uncontrollable vomiting, are almost surely fatal indications. Enteritis in different degrees of severity constitutes one of the most important causes of infantile mortality.

In the severer forms of enteritis, as they affect adults, opinion must be guided by the nature of the cause, and the general state of the patient. Recognising that the unfavourable tendencies of the disease are towards extreme prostration with fatal collapse, or to chronic ulceration, according as these conditions are threatened, so may the prognosis be fairly made. The duration of extreme cases rarely extends beyond a few days, when, if death do not occur from cardiac failure or pulmonary complication, the symptoms abate, and recovery, with oftentimes a tedious convalescence, follows, or a chronic condition of disease is established.

Treatment.—Although great variety exists in the degree of severity of the symptoms of acute enteritis, and a corresponding difference obtains in the treatment to be pursued, yet certain general principles may be first laid down, and the more special details adapted to certain conditions afterwards indicated. Inasmuch as the disease is one where the organs concerned with the preparation of the food are those mainly at fault, every effort should be made to minimise the bodily waste. This is best attained by keeping the patient in bed, which also offers the additional advantage of providing a uniform warmth.

As regards diet, in the greater number of cases of acute intestinal inflammation the appetite is much impaired, even to complete anorexia. Provided that the person attacked have been previously in good health, no harm is done by complete abstinence from food for twenty-four or even forty-eight hours. This gives a much better chance of rest to the intestine, and a better opportunity for the removal of any irritant ingesta which may have been the cause of the inflammation. The thirst during this period may be relieved by ice-cold water, with or without a little lemon-juice. It must not be forgotten that, with the mucous membrane and its glands inflamed, the conditions of normal digestion and absorption are materially interfered with, and articles of diet that ordinarily are most nutritious and easily digested may, and do become, under these altered circumstances, positively harmful. The aim in feeding the patient should be to give those materials which require the least digestion, and, being most quickly absorbed, leave the smallest amount of indigestible residue. If the stomach be implicated—and it is rare that it is not so—meat-foods are badly borne. Instead of the proteid constituents being digested, they undergo putrefactive decomposition, and thus add fresh irritants to the canal lower down. If, however, the stomach be tolerably free, then meat-essences, made thin and allowed to stand till cold, may be given. The nausea or vomiting which is usually present is more easily overcome by giving the nourishment cold; and a few drops of lemon-juice are of great service if added to the beef-tea. Milk is very uncertain in the way it is tolerated by such patients. Occasionally it is impossible to give it, the vomiting or diarrhœa being increased by it; but equal parts of milk and soda-water may constitute sufficient nourishment to last for several days in extreme cases, and may be well borne. Lime-water may be substituted for the soda-water; but, as a rule, effervescing fluids are more grateful, koumiss being often of great service. The milk should be sterilised and as free as possible from

cream, for fats in all forms are to be avoided, since the products of their decomposition are extremely irritating. Beef-tea or chicken-broth made with milk in place of water may be tried with advantage. Farinaceous substances, if given at all, should be administered only in small quantities at a time, a remark which equally applies to all other food. Thinly made Benger's food or plasmon is often most useful. Nutrient enemata are of value in some cases.

It is seldom advisable to check the diarrhœa in acute enteritis; and an aperient to begin with, unless any evidence exists of peritonitis, is a rational treatment; thereby the irritant, whatever it may be, is removed, and a better chance for recovery is given. Improper food is so commonly the cause, that the majority of cases are benefited by a preliminary purgation. Probably the best aperient in this case is castor oil, or calomel in doses of 1 to 4 grains according to age, but it may be necessary to follow up the latter after a few hours with a quickly acting saline aperient. The object is to clear out the alimentary canal; and provided that this has been done, abstinence from food for twelve hours, and some bismuth in an effervescing form, are frequently sufficient in milder cases to put the sufferer on the road to cure. The writer places great reliance on bismuth, either in the form of solution with an effervescing citrate of potassium, and 3 or 4 minims of diluted hydrocyanic acid; or granular effervescing lime-juice and bismuth. The nausea and vomiting are best relieved by this treatment. For the pain, poultices or poppy-head fomentations, or the internal administration of opium, may be very effective. Should there be peritonitis, the opium must be increased in amount, with a view to giving complete rest to the bowel. Several leeches applied to the anus, to relieve the hyperæmia of the intestinal tract, are occasionally necessary in extreme cases. When the attack is distinctly attributable to cold, a profuse sweating induced by hot baths, and 10 grains of Dover's powder, is often of great benefit. In those cases where, from the duration, character of stools, and previous treatment, there is reason to believe that the irritating causes are got rid of, the diarrhœa may then require special treatment, especially when the patient has been in ill-health, or is constitutionally debilitated. A powder, consisting of Pulv. ipecac. co. 5 grains, and Bismuthi carbonatis 10 grains, given every six hours, is very efficacious. Vegetable astringents are frequently used for the same purpose.

When the more acute symptoms have subsided, the bismuth may be still continued, and later on given with a vegetable bitter,

such as calumba. Ten to fifteen minims of the diluted hydrochloric acid in an ounce of water much assist recovery of the digestive power of the stomach, for which purpose also pepsin is valuable.

In consequence of the great liability to a second attack which this disease engenders, avoidance of well-known harmful articles of diet, and the use of warm clothing or flannel belts, are demanded as a prophylaxis.

In infants and young children the great liability to collapse, often rapidly fatal, must be borne in mind. Stimulants in some form are almost a necessity. The following prescription may be employed: Liquoris bismuthi et Ammonii citratis ℥j.–℥iij., Spiritus ammoniæ aromatici ℥ij.–℥v., Tincturæ cardamomi compositæ ℥ij.–℥v.; Aquæ ʒj.–ʒij.—according to age. Brandy in small quantities is often the means of saving life in these cases. When the collapse is not threatening, one or two drops of the B.P. solution of corrosive sublimate, with half a fluid-drachm of syrup, and a fluid-drachm and a half of water, every two or three hours, may be of great service. It is a more convenient mode of giving mercury than in the form of gray powder. But in one form or another the writer believes mercury to be of prime necessity. Corrections of diet on the lines indicated above are of course essential. Hot baths and other means to keep the child warm must be employed.

In the severer cases, which are lapsing into the typhoid state, the general principles for that condition must be followed; but, except in such a state, alcoholic stimulants are rarely called for.

Inasmuch as there is good reason to believe that many cases of intestinal inflammation, especially those of an epidemic character, are due to the activity of micro-organisms, the treatment of the disease on antiseptic principles has naturally been suggested. The problem has been to find a germicide which should pass to the ileum and colon without itself undergoing decomposition in the mouth, stomach, or upper part of the intestine, and should at the same time be harmless to the patient. Many are the agents which have been proposed, and with varying success. Few, if any, are superior to calomel or corrosive sublimate, or the solid preparations of bismuth. Thymol and salol are occasionally useful, but these latter, like resorcin, salicylic acid, salicylate and benzoate of sodium, salicylate of bismuth and cerium, the sulpho-carbolates of sodium or zinc, creosote, naphthalin, iodoform, and peppermint, are far more efficacious in checking fermentation in the stomach and upper bowel. Whichever remedy be employed, it should be given in frequently repeated small doses according to age. Irrigation of the

bowel by copious enemata of water, containing small proportions of one or other of the above-mentioned substances, is an effective method of intestinal antisepsis (see page 65).

Chronic Enteritis

Symptoms.—It is not always easy to say exactly when an acute case has lapsed into a chronic state, very much the same symptoms being continued. In such affections of the small intestine, the diarrhœa may be wholly wanting, and the bowels may be very confined. This is due to the diminished peristalsis, from impaired irritability of the muscular coat. When, however, ulceration is extreme, and especially if it be the colon or rectum that is mainly affected, chronic diarrhœa is an invariable symptom. The remarks made on the character of the stools in acute enteritis are equally applicable to the chronic state, with the addition, that solid and liquid evacuations frequently alternate. Lasting as the disease often does for many months or even years, a general impairment of nutrition results. The functions concerned in the elaboration of the food, as well as those by which the digested products are absorbed, are necessarily perverted. The marasmus is speedily noticed in infants and children, whose growing tissues less readily withstand malnutrition. Apart from the general ill-health produced, the mental qualities become affected, so that the intellect may become dulled and sluggish, the temper irritable, and the patient may fall into a condition of marked hypochondriasis. The emaciated appearance; the dirty muddy complexion, complicated often with a short dry cough, dependent on reflex irritation from the stomach, frequently lead a superficial observer to suspect the existence of phthisis.

Prognosis.—Chronic enteritis almost invariably tends towards a fatal termination, though this may be long delayed. The general nutrition becomes more and more deranged; and death from inanition finally terminates an existence of prolonged suffering and discomfort.

Treatment.—Owing to the unfavourable tendency of this disease, the treatment can be rarely more than palliative. The debilitating and wearying character of the malady emphatically calls for good feeding. The diet should be abundant and nutritious. But the giving of food is often difficult owing to the vomiting and pain produced; in such cases koumiss is of great value. In those

cases where the enteritis is a sequel of a congestion of the intestine, the treatment must be directed to relieve if possible the cause of that congestion. Since this is usually some such intractable condition as Bright's disease, cardiac dilatation, or cirrhosis of the liver, attention should be directed to the relief of these affections.

Tonics—such as quinine, iron, bark, with sea-air—are of undoubted benefit; and, so far as possible, causes of mental worry should be removed. Massage of the abdomen and gentle movements specially directed to the exercise of the abdominal muscles are most beneficial.

Catarrhal Colitis

In a large number of cases the colon is involved to a greater or less extent with the small intestine in the catarrhal state—entero-colitis—but not infrequently the morbid condition is restricted to the large bowel, and even perhaps to special regions such as the cæcum, sigmoid, or rectum. Although an inflammation of the mucosa of the large intestine, and to a somewhat less degree that of the small intestine, is very likely to proceed to ulceration, a colitis that is not so complicated is to be met with and will be here described, the symptoms referable to the ulcerative process being detailed subsequently. (For Morbid Anatomy, *vide* pp. 156, 161.)

With a common anatomical basis, viz. that of a catarrh, there is included within the clinical range of the term "simple or catarrhal colitis" a large number of cases which offer among themselves marked distinctive features. Thus some, attributable chiefly to dietetic errors, to irritant ingesta, or to the very real though imperfectly understood influence of "chill," run an acute course; others, associated with constipation and the constant presence of scybala, are subacute, and even more are chronic, and may appear to be so from the first; some also are characterised by definite attacks with irregular intervals of more or less complete freedom, and with no obvious cause for the recurrence.

Among the rarer conditions with which the malady has been found associated are Bright's disease, septic and pyæmic states, acute pneumonia, and malignant growths of the large intestine.

Some writers have considered that a close connection exists between catarrh of the colon and gout, or the so-called "uric acid diathesis." It is possible of course that an excretion of certain abnormal products of nitrogenous waste may take place by the

mucosa of the colon, and a catarrh be set up as the result thereof; but until more is known precisely of the nature of nitrogenous metabolism and the removal of the results, this can only be regarded as theoretical.

Other observers, especially of the French school, have been led to associate some forms of simple colitis with the "arthritic diathesis" and have not hesitated to include the intestinal symptoms within the manifestations of "arthritism." Here again more knowledge is wanted. That the joints are closely connected with the elimination of bacterial and autogenetic poisons appears to be certain, but the connection of the arthritic changes with catarrh of the colon, whether as cause, result, or coincidence, does not seem to the writer to be so evident. It is the cæcum and appendix that have been regarded as prone to an inflammation of a rheumatic nature, and the large amount of lymphoid tissue found in the latter structure has led to a comparison of it with the tonsils, and further of tonsillitis, which is so closely associated with acute rheumatism, with attacks of appendicitis. This can so far be only regarded as an interesting suggestion. It is, of course, possible that the bowel affection may occur in the subjects of acute rheumatism, though a pathological relation is not thereby to be assumed, and so far has not been proved. A polyarthritis developing in the course of perityphlitis has been noticed by several observers, but Dr. Poynton gives good reason for regarding such joint lesions as pyæmic rather than rheumatic (*Tr. Med. Soc. Lond.* 1901). And, lastly, the benefit that certainly seems to follow in some cases of the malady from the administration of salicylates, and has been claimed as evidence of the rheumatic origin of the attacks by some, and of their gouty nature by others, is not conclusive. Reference has already been made to the view that that form of colon affection which is distinguished by excessive discharge of mucus accompanied by considerable pain is essentially a secretory neurosis (p. 237). Objection has been taken to this view by those who regard the intestinal disease as determining a general neurosis by the depressing effect it exerts on the nervous system.[1]

However probable it may be that catarrhal colitis is an expression of bacterial invasion, there is no evidence at present to show that it is so, although various micro-organisms, including the B. coli communis and amœbæ, are found in the stools.

Symptomatology.—Great differences exist in the severity of

[1] "On Colon Catarrh," by Dr. T. Stacey Wilson, *Brit. Med. Journ.*, 6th Dec. 1902.

individual symptoms in different cases, though all present certain features in common. The most constant, as it is the most characteristic, manifestation is diarrhœa, with which the attack commences, and this continues the most prominent and may be almost the sole symptom throughout. The onset is oftener than not sudden. The most striking feature of the *stools* is the amount of mucus, offering therein a marked contrast to catarrh of the small intestine. Some loose fæculent matter, or it may be scybala coated with mucus, are voided, but the mucus is not intimately mixed with the fæces, remaining quite separate, whether in small discrete blobs or forming considerable clear jelly-like masses, or presenting the appearance of boiled sago, colourless, or sometimes stained with the fæcal colouring matter, or even greenish. In severe cases, or in the special form designated "mucous colitis," the motion may consist of almost pure mucus. Even more frequently, and in the more chronic cases, the mucus voided is of an inspissated, firmer character, with less of the clear jelly-like appearance. Such material appears as shreds, or flakes, or irregular masses, which on floating out in water are seen to be long, stringy or twisted membranous pieces of many inches or even feet in length, sometimes hollow and forming casts of the surface of the bowel—membranous colitis (see p. 161). Except perhaps some of the cases of mucous colitis, the symptoms of which may only recur at long intervals and are characterised by pain and pure mucous stools only, the others may be regarded as only differences in degree of one and the same disease. In the same case, if at all prolonged, mucus in the various forms described may be met with at different times. Bright blood in variable amount is often seen, but unless the condition be due to some violent irritant, its presence may be generally taken to indicate the presence of ulceration. Crystals of triple phosphates, oxalates, cholesterin, and Charcot's crystals are also to be found. The stools in mucous colitis often contain the so-called "intestinal sand," which bears a superficial resemblance to grains of uric acid. It chiefly consists of oxide and phosphate of lime, with traces of urobilin, and is undoubtedly formed in the intestine. It is to be distinguished from vegetable debris and particles of cellulose coated with lime salts. Notwithstanding that there is a frequent mucous diarrhœa, there may be actual constipation so far as fæcal matter is concerned, and this may be so considerable as to require measures for its relief. Marked irregularity of the action of the bowels is a characteristic feature. It is seldom that the diarrhœa is unaccompanied by pain, which commonly precedes defæcation and

particularly the passage of the "membranes," and though with no constant reference to food, not infrequently comes on about an hour after a meal, as it may after exercise. In all but the mildest cases it constitutes a distressing symptom, and is commonly described as griping and colicky, and may be agonising in its severity. It is felt generally over the abdomen, or confined to one region of the colon, as the sigmoid or cæcum, the course of the bowel being marked out by tenderness. When the case has lasted some time the patient is seldom free from some discomfort, though with paroxysmal exacerbations, very often at night and commonly increased by jolting movements, such as a carriage drive. During the paroxysm, and also in the intervals, a section of the gut may be felt to swell up and harden. Tenesmus occurs in many cases and irritability of the bladder is complained of when the rectum is involved.

Other symptoms referable to the alimentary system, though with no special constancy, are nausea, vomiting, anorexia, thirst, and a furred, or a bare red and glazed tongue. Not a few patients are confirmed dyspeptics, and manifest all forms of dietetic fads in the pursuance of their self-imposed rules of treatment. The gastric symptoms are sometimes very prominent, but when the transverse colon is specially involved the stomach is often erroneously supposed to be the seat of the mischief.

There is a slight rise of temperature in the acute cases, but the general symptoms of fever are scarcely present. Attacks of uric acid gravel have been noticed.

As is so commonly the case with affections of the large intestine, and perhaps especially in the disease now under consideration, there is a remarkable tendency to mental depression, amounting sometimes to hypochondriasis. This is to be met with in both men and women, though the latter, on the whole, furnish the majority of cases of the disease. The explanation is uncertain, but it is probable that the absorption of toxic material, due in part to the retention of fæcal matter, is largely responsible. In other cases a state of neurasthenia supervenes, and this perhaps is also more prevalent in females, who often become chronic invalids. Inasmuch as the subjects of the malady of either sex are distinctly neurotic, often with a "nervous" family history, it is not unlikely that these symptoms are rather to be regarded as being predisposed to, the disease acting as the exciting cause, and they have not infrequently been noted as occurring before the appearance of the bowel affection. In all a liability to cardiac depression exists, greater in the acute cases,

which adds to the general enfeeblement which the more advanced cases exhibit, though any great emaciation is often wanting, since the upper part of the digestive tract may satisfactorily deal with the food taken. When, however, the pain is great, and sleep is interfered with, or has to be induced by drugs, then the general nutrition will probably suffer.

Painless vomiting and gastric hypersecretion have been noted among symptoms of reflex causation (Dr. T. S. Wilson, *loc. cit.*), and the writer has noticed in more than one case a tendency for other mucous membranes to assume a catarrhal state, especially the uterine, which is further complicated by dysmenorrhœa.

The limitation of the catarrhal condition to the cæcum—*catarrhal typhlitis*—has been mentioned, and its clinical features appear to be tolerably definite, though the presence of ulceration cannot be always excluded. Such cases have been included within the somewhat heterogenous collection known as perityphlitis or appendicitis (see p. 352), but as the greater number recover the precise anatomical condition remains doubtful. The attack arises somewhat acutely in persons of constipated habit, in the writer's experience oftener in females, and is commonly induced by a heavy meal, especially if this contain much indigestible matter. Considerable pain, dull with exacerbations, is complained of in the cæcal region, which is swollen, tender, resistant, and dull on percussion, the prominence being generally well defined and due to fæcal accumulation and the thickened wall of the inflamed bowel. The temperature is raised and runs an irregular course, reaching 103° F., or even higher; the pulse is soft and quick. There is marked constipation, and frequently nausea and some vomiting. The patient lies on her back, and often draws up the right leg and flexes it across the other. The symptoms may last for days, even ten or more, and are subject to considerable fluctuation in severity from day to day, the limits of the abdominal swelling exhibiting a similar variation. The majority of such cases clear up, and recovery is complete, although recurrence may take place; but now and then one proceeds to suppuration, and it is probable that in such the appendix is involved, leading to localised peritonitis, though a stercoral cæcal ulcer may occasionally be the cause of the perityphlitis.

Although doubtless the appendix is frequently the seat of a catarrh, whether by extension from the cæcum or primarily, it cannot be said that a *catarrhal appendicitis* is clinically recognisable, and in such cases which have given rise to pain—so-called appendicular colic—or have proceeded to peritonitis, there has been some

erosion or ulceration of the mucosa. The structure has sometimes been found much thickened, with more or less complete obliteration of its lumen, and presenting the anatomical features of a chronic catarrh.

Prognosis.—Although the mildest cases recover, and sometimes quickly, the disease is one that should always be regarded with some anxiety from the facility with which it becomes chronic and the liability to assume an obstinate and intractable form. Even when cure seems to have been obtained a relapse readily happens, and the longer the condition has existed the less chance is there for complete recovery.

Treatment.—It is most important that the food of patients suffering from this complaint should be as free as possible from indigestible residue, such as vegetable fibres, husks, seeds, etc. In other respects the diet should be generous, and certainly not confined to liquid foods; although cases do occur in which more pain and discomfort are experienced from solids, and in such an exception must be made. Alcoholic stimulants should as a rule be withheld. The general health, and especially the liability to mental depression, must be carefully looked after by general hygienic and tonic treatment, among which strychnine, arsenic, and iron in some form are the most valuable means, and such social and climatic measures as may be possible, particularly avoiding cold and damp situations. The clothing should be warm, and much comfort is to be derived from a simple flannel belt, which should be worn during the day, and great relief follows the application of a layer of heated wadding covered with oil-silk and kept in place by a flannel belly-band. Hot fomentations may be necessary if the pain be very severe, or even small doses of Dover's powder. Locally great benefit is derived from the administration of salol in full doses (5 to 8 grs., three or four times daily); watching the state of the urine. It is commonly the case that some laxative is required, and a daily dose of castor oil, if necessary in capsule, is the most efficient. Saline aperients may be given, or even small doses of cascara sagrada or podophyllin with belladonna. Dram doses of the confection of bael have been recommended for the relief of the pain and constipation.

Irrigation of the bowel in the more chronic cases is often most beneficial; using warm saturated solutions of boric acid or of liquor ferri pernitratis (℥j. to ℥ij. to the pint), with the addition of a little of the liquid extract of opium if the fluid cause much pain. Other substances, such as nitrate of silver, solutions of bismuth, ichthyol, and naphthol have been recommended for administration in the same

manner. In all cases it is essential that the irrigation be practised slowly, several pints being gradually and successively introduced, so as to wash the surface of the bowel as much as possible, the patient being turned from side to side to allow the fluid to follow the course of the colon. At certain watering places, notably at Plombières and Royat, this treatment is very successfully carried out, the natural waters being used for the purpose, and also taken internally.

In very intractable cases Dr. Hale White and Mr. Golding-Bird (*Tr. Clin. Soc. Lond.*, 1899 and 1902) have performed right lumbar colotomy, with the object of giving the colon absolute rest, the artificial anus being subsequently (after several years) closed, with complete cure of the disease.

When the morbid condition is confined to the cæcum—typhlitis—the benefit from a course of salicylates should not be forgotten, but when, as is much more commonly the case, fæcal accumulation is the underlying condition, steps must be taken to unload the bowels. A daily simple enema is the best evacuant, warm olive oil appearing to give considerable relief to the pain as well as emptying the colon, and it is most important that no violent purgation be attempted; a dose of a saline aperient once or even oftener in the twenty-four hours may be desirable with a view to soften the fæces. The patient will of herself keep her bed, induced thereto by the pain and sense of illness. Hot fomentations, with applications of extract of belladonna over the cæcal region, or a few leeches in the same situation, may be required for the pain, which is best allayed, as well as the restlessness soothed, by small doses of Dover's powder, which should only be just sufficient for this purpose. The writer strongly deprecates the routine withholding of opiates in such cases from a fear of "masking the symptoms"; it is quite possible to give the much needed relief without obscuring the course of the case. Food should not be pressed, certainly not at first, a little fluid nourishment, milk, or beef-tea, is quite sufficient for some days, whilst the anorexia and nausea make feeding difficult.

ULCERATION OF THE STOMACH AND BOWELS

Ulcer of the Stomach

For the morbid anatomy and pathogenesis of gastric ulcer see p. 170.

The **symptoms** of a typical case of gastric ulcer are few in

number and precise in character, presenting a clinical picture concerning the nature of which there can be little mistake. But in the consideration of all cases which may fairly and properly be comprised within this category, very considerable exceptions have to be made, and no one of the symptoms, even of those most definite, is of invariable occurrence. Severe epigastric pain, much intensified, or called forth by food and unrelieved until the stomach be emptied of its contents by vomiting or by propulsion through the pylorus, with a more or less profuse hæmatemesis, occurring in a young person, especially a female, constitute the leading features upon which a diagnosis would be made without much hesitation. There exist, however, great differences in the general condition of the individuals in whom such symptoms may occur, in the subsequent course and complications which may arise, and, so far as is known, in the character of the gastric lesion; so much so, that whilst closely corresponding in the essential manifestations, patients suffering from what is with good reason recognised as ulcer of the stomach nevertheless are widely contrasted in the entire clinical characters of their disease.

Pain.—Of the three leading symptoms—pain, vomiting, and hæmatemesis—pain is the most constant, and in cases of extremely rapid course which terminate by perforation it may be the only one, to be followed at once by collapse and other evidences of peritoneal involvement. Sometimes the pain is almost or quite continuous, and sufficiently intense to interfere with sleep, but far more often there are periods of relief, a paroxysm being excited by the food that is swallowed, setting in within a few minutes to half an hour or more afterwards. Some attempts have been made to locate the situation of the lesion by the time that elapses between the taking of food and the onset of pain, the longer the period the nearer to the pylorus; but since the pain is often partly of a neuralgic nature, being induced by other conditions, such as exposure to cold, excitement, exertion, or even by menstruation, the result is not altogether satisfactory. The character of the pain is variously described as burning, boring and shooting through to the back, sometimes so severe as to cause fainting, and tending to become more intense as the case progresses. As a rule, it is strictly localised, and the patient points to a spot just below the ensiform cartilage, sometimes as well, or it may be only, at a spot in the back immediately to the left of the tenth or eleventh dorsal vertebra. Occasionally the pain radiates from the epigastric region, or like so many pains of gastro-intestinal origin, may be felt about the

umbilicus, or be referred to a higher point along the spine between the sixth and eighth dorsal vertebræ, or even to either shoulder or arm, or along the intercostal spaces. How far these variable situations are connected with the actual site of the ulcer is uncertain, although some indication of this may sometimes be obtained by noting variations in the degree of pain that follow alterations in the position of the patient; thus when relief succeeds to lying on the face, the ulcer may be on the posterior wall, or at the cardiac end when the right decubitus is assumed. Pressure over the epigastrium almost always increases the pain, and the tenderness and hyperæsthesia in this region may be so great that even contact with the bedclothes is unbearable.

Such are the usual characters, but to each one of the statements exceptions occur. Thus, pain may be wholly wanting. Large ulcers have been found in the stomach post-mortem, which have given rise to no symptoms during life, and whose existence was quite unsuspected; or it may be that a violent hæmatemesis has first suggested the lesion, and a painless recovery has ensued; or pain may cease for long periods to recur later, whilst other evidences of ulcer persist. Or, again, the pain may have no such definite relation to food as here described; it may come on when the stomach is empty long after a meal, or it may be almost continuous, and but little, if at all, influenced by what is swallowed, the explanation of which is doubtful. A flow of gastric juice of increased acidity has been suggested to account for the former phenomenon, and this may be why food has very rarely been known to relieve the discomfort. The permanently distended stomach may determine a constant pain, and the associated gastritis may be partly responsible. Very exceptionally, pressure over the epigastrium has been known to afford relief, and in a case of duodenal ulcer within the writer's knowledge which was fatal by perforation, this circumstance was very marked.

Vomiting.—This is almost if not quite as constant a symptom as pain, being very seldom absent when the ulcer is in the usual situation, viz. on the posterior wall at the pyloric end, although when the lesion is on the anterior surface or at the fundus it may not occur. Like the pain, it is usually induced by food or by an accumulation of saliva that has been swallowed, which act as irritants to the exposed nerves; at the same time it is also liable to be excited by mental or bodily exertion, or other circumstances acting reflexly. It may follow almost directly on taking food, or not for an hour or more. As a general rule the pain is relieved,

perhaps entirely, by the vomiting, although exceptions to this do occur. Free hydrochloric acid is always present in the vomit, very often in excess, together with the products of digestion, albumose, etc., and frequently lactic acid. It is, however, the presence of blood, usually in large quantity, from several ounces up to one or even two pints, that especially characterises the ejecta. The *hæmatemesis* may occur only once or be repeated twice or more within a day or two, and is not infrequently preceded by an accession of pain, or by peculiar sensations of heaviness or distension of the stomach. The blood is bright, and coagulates after being voided, or clots may be brought up. However profuse the hæmorrhage may be, it is comparatively seldom fatal, though such a result is not unknown, and death has followed an escape of blood into the stomach without any being vomited or passed per anum. The bleeding, of course, reduces the patient to an extreme degree of faintness, and this, as well as pallor, are proportionate to the loss of blood. In exceptional cases hæmatemesis is the first symptom to suggest the presence of an ulcer in a patient who has made no previous complaint of either pain or vomiting, or even of the mildest dyspeptic troubles. Further particulars concerning this symptom and the consequent *melæna* are given on p. 238.

Investigations directed to the functional capacity of the stomach, when the seat of an ulcer, have demonstrated a very constant excess in the secretion of hydrochloric acid, which has previously been referred to as probably playing some part in the production of the lesion. This hyperchlorhydria may even be met with when the stomach is empty, and in the dilatation which may follow on pyloric stenosis caused by cicatrisation of the ulcer. The pepsin also would appear to be frequently increased. Some deficiency of gastric motility is apt to occur when the general health is much impaired, as in chlorosis or in gastroectasis; and such absorption as takes place from the stomach is alleged to be delayed in this disease.

Associated with these very definite manifestations, there is a variable degree of gastric dyspepsia, which may be due to a chronic gastritis of some duration, in connection with which the ulcer may have developed; or the indigestion may have been of the nervous variety and closely connected with secretory or sensori-motor disturbances in the stomach. Since, also, the greater proportion of cases classed as gastric ulcer occur in young and chlorotic women, the failures in normal gastric digestion as well as many of the general symptoms may be properly referred to the quality of the blood and the malnutrition dependent thereon. Thus it is that the

appetite is commonly very indifferent or capricious, and thirst is complained of, especially if there have been any hæmorrhage; the tongue is pale and flabby and indented by the teeth; and constipation, due to the deteriorated tone of the intestinal musculature and the lack of adequate stimulus to peristalsis from the small bulk of the contents, is the rule. With these are various degrees of discomfort, perhaps amounting to pain in the stomach or abdomen generally, with a sense of fulness and sinking. But such symptoms as these by no means necessarily occur, and they are not peculiar to nor even essential manifestations of ulceration. They may have existed for long previous to those due to the ulcer, or they may set in when those caused by the specific lesion have in great measure passed away, or they may be wholly absent throughout.

Other evidences of perverted function which may be met with in the course of gastric ulcer are probably only remotely related to that condition, and are rather the secondary expressions of disturbance, or result from the general state of bad health accompanying the special stomach affection. The temperature, apart from any inflammatory complication, is normal or even below. The heart, which is generally undisturbed, may become irregular and develop various murmurs when the anæmia is considerable; from lack of sufficient nourishment the force and tone of the circulation is likely to be depressed. To the general state of ill-health and dyspepsia may also be ascribed such specially nervous phenomena as headache, vertigo, and sleeplessness. Dr. Dreschfeld records three cases in which general convulsions occurred, probably in connection with hæmorrhage, and the same observer also saw tetany in one case, occurring independently of washing out the stomach. The subjects of the malady being most frequently young women, the symptoms of neurasthenia or of hysteria are not unlikely to coexist. The *urine* may show some abnormalities due to the altered gastric state; the hypersecretion of hydrochloric acid may considerably reduce the acidity of the renal excretion, in which also the chlorides may be diminished and the phosphates increased. Both albuminuria and albumosuria may occur, and glycosuria is not unknown; indican and ethereal sulphates bear witness when in excess to unhealthy bacterial fermentations in the intestines. Amenorrhœa is a very constant occurrence in the chlorotic young women.

Course and Termination.—Within the limits of the foregoing account almost every case of gastric ulcer would find a place; it is in proceeding to consider the course and termination of the disease that marked differences of type are disclosed.

(*a*) *Recovery.*—By far the greater number of cases of what is a far from uncommon affection get well, and this is pre-eminently so when the subjects are chlorotic young women who form the greater proportion. Coming on in a patient profoundly anæmic, and with a vitality and capability much impaired therefrom, or supervening suddenly in a girl who is only just able to drag through her day's work, she is in a very short time reduced to an alarming state of prostration from loss of blood, which, as said, may recur on two or three days. Yet with care recovery almost always follows, though the patient is left more anæmic than before, but with no specially marked gastric symptoms either of pain or vomiting. In a few months or a year these indications set in again, and another hæmorrhage takes place, again to be followed by recovery, and this may be again repeated, or the first outburst may be the only one. It is important to notice that a very large number of young women, especially of the servant class, are admitted to hospital suffering from repeated attacks of great pain and vomiting and all the recognised symptoms of gastric ulcer, except that there is no hæmorrhage, and it is customary to include, at least the severest of them, as gastric ulcer, thus helping to swell the preponderance of the disease in females. Some observers, however, hesitate to diagnose ulcer unless there be a loss of blood, and have been satisfied to designate such cases as gastrodynia. The special feature here insisted on is that the greater number, whether there be bleeding or not, recover, and many ultimately become perfectly healthy women as the chlorosis disappears. The recently introduced practice of treating gastric hæmorrhage by opening the stomach and ligaturing the bleeding vessel has given the opportunity for the exploration of the viscus after hæmatemesis, and in a certain number of such cases no ulcer has been found, and nothing more than a minute erosion of the mucosa. This fact has shown that a definite ulcer such as previously described is by no means necessary for the production of the intense pain, vomiting, and hæmorrhage, and it also suggests that it is not the actual lesion itself, but rather perhaps some general sensitiveness of the gastric mucosa connected with the general state of the patient, which may or may not be associated with bleeding according as the erosions happen to involve a vessel of any size, that chiefly accounts for the pain and vomiting. This would bring the bleeding and non-bleeding cases, as they occur in chlorotic girls be it understood, within the same category, the hæmorrhage being, so to say, an accidental complication.

(*b*) *Perforation.*—Whilst the greater number of cases run an

uncomplicated course towards ultimate recovery, a certain proportion terminate in this serious and usually fatal event. No indications exist that foreshadow such a result, and the clinical course of the case up to the occurrence of perforation may be in nowise different from those which recover. Post-mortem, however, as might be expected, a very definite ulcer extending through the several coats is discovered, presenting all the anatomical characters previously described. Inasmuch, however, as the "peptic" or "perforating" ulcer, there is reason to suppose, starts from a simple erosion, the difference in the clinical as in the anatomical characters of these cases is only one of degree. As with hæmorrhage, so with perforation, it may be the first symptom that calls attention to the condition, and from a state of apparently sound health the individual rapidly passes into one of extreme danger. Such cases as these, in which these grave symptoms suddenly appear without previous discomfort sufficient to warrant complaint, have been termed "latent," a designation, it may be observed, which is still more applicable to those ulcers found after death, whose presence has been quite unsuspected during life, having given rise to no recognisable symptoms whatever.

The actual proportion of cases of ulcer of the stomach which end in perforation is hard to determine, owing to the doubt as to what is exactly included under this name; probably it does not exceed 10 or 12 per cent. A curious difference has been noticed as regards the age at which this complication occurs in the two sexes, the average in women being between twenty-five and thirty, and in men between forty and forty-five.

The symptoms indicative of this accident are indistinguishable from those attending perforation into the peritoneum of the hollow viscera generally. There is commonly an attack of acute and agonising pain, not necessarily felt at the epigastrium, quite as often at the umbilicus or even diffused over the abdomen, or occasionally over the region of the cæcum, suggesting perforation of the appendix. The patient rapidly falls into a state of collapse, the temperature falls, the surface and extremities are cold and bathed in a clammy sweat, the heart's action is greatly reduced in force, and the radial pulse may not be felt, or is quick, small, and thready, the breathing is shallow and hurried, and consciousness remains. Unless death take place, peritonitis begins to be manifested within twenty-four hours, and to this the fatal end may be due, unless operative interference should have been successful (see p. 245).

When perforations have occurred on the posterior surface of the

organ, and more particularly if there have been any adhesions previously set up by the extending ulcer, death is avoided, and an abscess of a short but more commonly of some considerable duration is formed in the post-peritoneal connective tissue—sub-phrenic abscess—though this is said to more usually follow a duodenal than a gastric lesion (see p. 520).

(*c*) *Cicatrisation.*—The healing of a gastric ulcer is known rather by post-mortem observation than by any special manifestations during life, and this is particularly so since, though the lesion be healed, many of the symptoms are by no means necessarily removed. Pain after food may continue, whether from the scar being irritable or from the interference to the peristalsis offered by the interposition in the muscular coat of a band of fibrous tissue, or it may be from adhesions which have been developed externally to adjacent parts. The vomiting may also continue, and this with the pain may maintain the general ill-health of the patient. Very many cases, however, do recover completely, and in such it is allowable to assume a satisfactory cicatrix has been formed.

The subsequent contraction of the scar is very liable to set up a fresh set of difficulties. If it have been in the neighbourhood of the pylorus, stenosis of the orifice, even to an extreme degree, may ensue, and lead to very considerable dilatation; or should the ulcer have been situated on the lesser curvature and extend over both surfaces of the stomach, an "hour-glass contraction" is very likely to be the result. Mention has previously been made of the possible relation of carcinoma to a gastric cicatrix (p. 182).

(*d*) *Chronic progressive ulceration.*—Some cases which have at the outset conformed to the general character and course of gastric ulcer, instead of recovering or of ending in perforation or cicatrisation, continue to exhibit symptoms more or less continuously, or perhaps only at intervals over many years. Here again it is in great measure what is found post-mortem that enables us to assign the case to its proper group. There is found after death an ulcer perhaps of considerable size—several inches in diameter—that has completely extended through the walls of the stomach, the base being formed by the pancreas, liver, or other adjacent structures. At the same time, it must be admitted that such a lesion has been met with that has given rise to few or no symptoms during life, and has run an entirely "latent" course. It would seem very probable that such a condition is due to firm adhesions having been developed in the progress of the ulceration, thus averting what would otherwise have been a perforation into the peritoneal cavity, and that these

same adhesions have at the same time prevented cicatrisation, with the result that the ulcer has slowly extended, finally to terminate by perforation or by erosion of a large vessel and fatal hæmorrhage. When this state of things has existed in the pyloric region, the chronic inflammatory process, giving rise to a considerable formation of fibrous tissue, comes to form a tumour which may require very careful consideration to distinguish from a malignant growth.

Diagnosis.—The recognition of the existence of ulcer of the stomach is generally easy. Special attention should be directed to the definitely localised character of the pain and tenderness, the relation of the pain to the taking of food and its relief by vomiting, to the occurrence of hæmatemesis, the presence in the vomit of an excess of hydrochloric acid, together with the age and sex of the individual, bearing in mind that exceptions to the general rule may be met with in respect to each of those conditions. According to the view taken as to how far simple erosions which, it has been demonstrated, may give rise to all the cardinal symptoms of ulcer, are to be included within the term ulcer, so will be the diagnosis of many milder cases in which perhaps the hæmorrhage is quite absent. To some of these cases the name gastralgia is possibly more applicable, especially since some of the pain and vomiting even with definite ulcer are probably due to a hyperæsthetic state of the gastric mucosa. If the pain be relieved by food the condition is most likely to be one of hyperchlorhydria. Much reliance may be placed on the strictly localised character of the pain and tenderness, for though this is not invariable it scarcely if ever characterises the pain of other gastric affections or of lesions of neighbouring parts. It is specially serviceable in helping to distinguish ulcer from cancer of the stomach, together with due consideration for the age of the patient, the excess of hydrochloric acid in the stomach contents in ulcer, and that the blood brought up is oftener "coffee-ground" than bright in character in cancer, and is seen late in the disease and not at the outset. But these features are less distinctive when the ulcer is one of long duration in a person over middle life, and the diagnosis becomes exceedingly difficult when an epigastric tumour is to be felt, such as a dense inflammatory thickening might give rise to. This is the more important as much benefit may result from operation in the one case, and harm rather than good in the other. It is needful to consider the whole bearings of any particular case, especially its duration, the mode of onset, the development of a cachexia or otherwise, as well as the special points of difference to which attention has been directed.

The differentiation of gastric from duodenal ulcer will be more conveniently referred to under the description of the latter condition.

Prognosis.—The uncertainty of the course and termination in any case presenting the symptoms of gastric ulcer will be apparent from the foregoing account. The greater number recover completely, although there may be recurrence before good health is fully established; very many exhibit symptoms more or less severe for many years, and finally succumb to one or other of the complications which may be associated with the ulcer or even follow on its healing; and lastly, a minority die within a short time, days or weeks, from the recognised onset of the disease. Statistics of these several groups are very various, and afford but little help in forecasting the probable course of any particular case. It has been estimated that 5 per cent of the entire population suffer from ulcer of the stomach, and that in 80 per cent of these hæmorrhage occurs, about 7 per cent of which are fatal, and in nearly 3 per cent death is due to perforation. But too much importance must not be attached to these figures. Of the proportion of recovered cases, and of those which remain chronic invalids, statistics are wanting. The most definite assertion that can be made is that the severest case of hæmorrhage may recover, and the apparently mildest case may be rapidly fatal from perforation, though surgery is doing much to reduce the risks of both these accidents. The deteriorated health induced by the malady may favour the invasion of tuberculosis.

Duodenal Ulcer

Although of commoner occurrence than is usually supposed, this affection is less frequent than ulcer of the stomach, which it closely resembles in its clinical features, as it also corresponds in anatomical characters and pathogeny (*vide* p. 173). Mention has been made of the singular difference in respect to age and sex incidence; and the disease is far less frequently associated with chlorosis than the stomach affection.

Symptoms.—As with gastric ulcer, not a few cases exhibit no symptoms until the occurrence of a fatal hæmorrhage, or more often perforation, which is followed by death within twenty-four or forty-eight hours; and the frequency of these "latent" cases is further demonstrated by the not uncommon occurrence of cicatrices in the duodenum found post-mortem, with no history of any symptoms

pointing to the existence of an ulcer during life. Sometimes, no doubt, such cases have exhibited vague and indefinite dyspeptic trouble, pain, discomfort, a sense of fulness, impaired appetite, etc., which have been attributed to a mild, subacute, or chronic gastritis, and have been paid but little attention to, and in the absence of more definite indications it could not well have been otherwise.

When, however, symptoms are well marked they follow, as said, those manifested by a gastric ulcer, with such differences as serve as grounds, often far from certain, for a differential diagnosis.

Pain, which exhibits all varieties of character and severity, from a very moderate dull ache to being severe and agonising, is less constant in its occurrence than when the stomach is affected, and as a rule is experienced one to three hours after food is taken. It is seldom very definitely localised in situation, and is felt more generally over the right hypochondrium rather than in the epigastrium, extending into the right loin, and is certainly less often referred to the back, though it may be felt in the right shoulder. Pressure over the painful region may increase the discomfort, or the reverse. *Vomiting* is much less frequent than in gastric ulcer, nor does it afford any special relief to the pain, and like that symptom occurs several hours after food. It may be altogether absent, or only occur once or twice in the course of the malady, hence its diagnostic value is of much less importance. The vomit may contain traces of altered blood, possibly also of bile, and there is said to be no such constant excess of hydrochloric acid as is met with in the stomach affection. *Hæmatemesis* also occurs much less often, and only when a very considerable hæmorrhage takes place. *Melæna*, on the contrary, is more frequent, and takes the place, as it were, of hæmatemesis, which it often precedes over some period.

The general malnutrition which such a disturbance of the digestive process engenders is shown by the wasting and muscular weakness, as also by the state of the blood, which in this as in gastric ulcer comes to resemble that characteristic of pernicious anæmia, and is still more marked in cancer of the stomach, a circumstance which lends support to the view that the essential cause of this grave blood disease is some autogenetic toxin developed in the gastro-intestinal tract. The bowels are apt to be confined, or this state may alternate with diarrhœa. An associated catarrh of the duodenum may determine a moderate jaundice.

Prognosis.—Death may be due to hæmorrhage, but oftener to perforation, and occasionally from progressive malnutrition. A fair number of cases, in whom there is reason to suspect the existence

of this malady, may go on for some years, with exacerbations at intervals, which may be of long duration, and it is not unusual to meet with patients who give an account of what strongly suggests duodenal ulcer many years previously followed by almost complete relief with subsequent recurrence. Cicatrisation of the ulcer may lead to stenosis of the bowel, with all the troubles dependent thereon; or the orifices of the bile and pancreatic ducts may be involved in the scar, with the production of persistent and severe jaundice, and great emaciation, with fatty stools, etc.; or perforation, instead of being immediately fatal, as it most usually is, may give rise to a sub-phrenic abscess or establish a fistulous communication with the colon or gall-bladder. As in the stomach, the scar has been known to be the seat of subsequent cancer.

Diagnosis.—The data for distinguishing between a gastric and a duodenal ulcer have been sufficiently set forth in the account of the symptoms which the latter exhibits, bearing in mind the age and sex differences of their occurrence, as well as the greater frequency of a pre-existing chlorosis in the stomach lesion, and of an extensive skin burn in the duodenal. But the diagnosis is not so easy as might appear from a description that aims at giving a generally complete account of these diseases rather than contrasting individual cases, and when the duodenal ulcer is close to the pylorus distinction may be impossible. An absence of symptoms of gastric dyspepsia is said to be noteworthy in duodenal ulceration, but this is not to be distinguished with anything like certainty, since the bowel lesion does in varying degree interfere with the stomach functions.

With somewhat more reason than gastric ulcer the symptoms of the duodenal lesion might be mistaken for gall-stone colic. The pain is felt much in the same situation, and may be referred to the right shoulder in both; vomiting may occur in either condition; and jaundice, which is usual with biliary colic, may complicate the duodenal lesion. A rise of temperature and perhaps a rigor and sweating are in favour of gall-stones.

Acute pancreatitis is a rare condition, which may resemble duodenal ulcer in some of its symptoms, and lead colic may be mistaken for it.

Treatment.—This should aim at healing the ulcer, and at the same time relieving the symptoms to which it gives rise or that are connected with such conditions as may be present, viz. catarrh of the stomach, hyperchlorhydria, and chlorosis. To a great extent these several indications are met by similar lines of procedure.

When once the lesion has been diagnosed, whether subsequent to a hæmatemesis or not, and even when only reasonable probability exists for its occurrence, complete rest in bed should be insisted on, and should be maintained until well after all symptoms have ceased. By this means the risks attendant upon the malady are diminished, and the chances of complete healing and avoidance of a chronic intractable ulcer being established are increased.

Opinions differ as to the value of remedies that may be supposed to promote the healing by their direct effect upon the wound, such as nitrate of silver or subcarbonate of bismuth. By some, one or other of these substances is given regularly over some weeks, whilst others discard them as useless for the purpose assigned. The writer thinks benefit in this direction does follow a pill consisting of argenti nitratis gr. $\frac{1}{4}$ to $\frac{1}{2}$, sodii carbonatis exsic. gr. iij., kaolin *q.s.*, given two or three times daily.

The principle of rest should be still further applied by complete abstinence from food by the mouth, and feeding by nutrient enemata or suppositories. This is strictly to be followed if hæmorrhage have occurred. Thirst should be relieved by a teaspoonful of tepid water now and then and warm water enemata. It is very doubtful whether sucking lumps of ice, so often recommended, does not really favour the bleeding; and the stomach is apt to be burdened by a larger quantity of fluid in this way than is generally supposed. For how long rectal feeding should be continued depends on the ability of the patient to retain the injections and on the general condition, and also on the view of the case taken by the physician. Regularly washing out the rectum previous to the nutritive enema, and the addition thereto of a small quantity of opium, will allay the irritability of the bowel and permit a longer continuance of the treatment, which may in favourable cases be pursued for several weeks, though the patient loses weight very considerably, and this fact should enter into the considerations limiting its duration. But there is another aspect of rectal feeding which is of considerable importance, especially since it is a method of alimentation that should not be entered on, or at least maintained beyond a few days, except from necessity. Observation has shown that very many cases of chlorotic girls exhibiting all the symptoms of gastric ulcer, and many others in whom hæmorrhage is alone wanting, do very well on a fairly good diet of solid food, including meat, almost from the first manifestation of symptoms, and much sooner regain their health than those who are kept on nutritive enemata and suppositories for weeks. It has also been proved at operation that some of them are the subjects of

a minute ulcer or erosion, and it is probable that a very large number are suffering from a similar lesion. For such patients a full diet might reasonably be supposed to be innocuous, while the presence of a large ulcer that has penetrated through the mucosa to the muscular layer, or even deeper, might on equally good grounds be regarded as distinctly prohibitive. With the identity of symptoms in the two groups of cases the difficulty is to decide to which any individual one can be referred; the probability on the ground of frequency alone in favour of the former class being scarcely sufficient to define the regimen to be ordered. Intermediate, as it were, between these extremes of dietetic method may be mentioned a purely milk diet, very small quantities being given at frequent regular intervals, the amount being increased as improvement takes place. The milk may be peptonised, or mixed with lime-water or with effervescing water—soda, Apollinaris, etc.—or with a small quantity of farinaceous food, such as barley-water or oatmeal, all designed to cause a looser coagulation of the casein. Others prefer to give three or four meals a day of beef powder or some finely divided dry proteid mixed with magnesia or prepared chalk, and others depend on frequent teaspoonful doses of strong meat essences. Each of these methods the writer has seen attended with success, and on the whole it may be said that the general opinion is inclining rather to oral feeding, at least after two or three days' abstinence or rectal injections. As the case improves a more varied dietary may be gradually introduced.

Of the special manifestations, pain more particularly calls for treatment, and for this purpose the various gastric sedatives may be tried. But since this symptom, as well as nausea and other sensory discomforts, is closely associated with the excessive acid secretion in the stomach, the correction of this condition goes far to allay the discomfort. For this purpose the bicarbonates of soda or potash (gr. xxx.) are most effective, and may conveniently be given with subcarbonate of bismuth (gr. xx.) and oxalate of cerium (gr. x.) Or, acid. hydrocyan. dil. ℥ iij., liq. bismuthi et ammonii citratis ℥ xxx., sp. chloroformi ℥ v., aq. ad. ℥ ij. the writer has found most efficacious, and to this, if need be, three drops of liq. cocainæ hydrochlor., or of liq. morphinæ acetatis may be added. As a rule, however, unless the pain be very severe, or unless there be hæmorrhage, it is well to avoid opium and morphia. In the case of bleeding, their value is great in calming the patient, and in consequence favouring its arrest; morphia given subcutaneously is the most satisfactory form. Atropin (gr. $\frac{1}{100}$) may be given with a view to arrest the

secretion of the hydrochloric acid, being repeated every four or five hours according as it is tolerated. Hot fomentations also give great relief to the pain.

Constipation, which, as a rule, is extremely obstinate, must be overcome by enemata and laxatives, such as cascara, or, what is perhaps generally preferable, castor oil or Carlsbad salts. The vomiting is best treated by the measures just mentioned for the relief of pain, which may be supplemented by a sinapism applied over the stomach. If the hæmorrhage be very excessive, transfusion of normal saline solution should be at once resorted to, as this diminishes the risk of syncope and prevents the distressing thirst which is so likely to follow. Most of the ordinary hæmostatics, so called, are useless, but the administration of adrenalin chloride in a dose of 25 drops of a 1 in 1000 solution, or in 5-drop doses repeated every two hours, appears to be very successful. Half-dram doses of the solution of perchloride of iron are also strongly recommended; or the subcutaneous injection of ergotin, repeated every few hours for several doses, may be employed.

The importance of keeping the patient under treatment for some weeks after all symptoms have ceased, and under observation for some time longer, cannot be too strongly insisted upon. The subsequent anæmia supervening, as is generally the case in a state of chlorosis, requires iron, meat, sunlight, and the ordinary management pursued in this condition.

Considerable success has followed the radical treatment of the ulcer by excising it and suturing the wound, or by performing a gastro-jejunostomy, operations which are applicable for the hæmorrhage or for perforation. In all cases of healed gastric ulcer, except when the erosion is minute, there are the risks attendant upon the contraction of the cicatrix. These it is practically impossible to avert, but a constriction of the pylorus may be open to surgical treatment, either by dilating the orifice or by establishing a communication of the stomach with the jejunum.

Ulceration of the Jejunum and Ileum

Ulcers in the small intestine, excluding the duodenum, are most commonly of infective origin, typhoid or tuberculous, much less often catarrhal or follicular, and still more rarely the result of lardaceous degeneration or associated with Bright's disease (see p. 164).

Symptoms.—Few, if any, of the symptoms can be regarded as characteristic. The lesion may occur without producing any indications; or those that do exist may be determined by the course of, or by the circumstances associated with, the ulceration; or, lastly, the results of this condition, such as perforation, may entirely obscure the actual ulceration itself; it frequently happens therefore that the existence of an ulcer is not recognised. Nor may the severity of the symptoms be taken as a measure of the extent of the ulceration, for the most marked pain, tenderness, diarrhœa, and other symptoms may be produced by an area of typhoid ulceration that heals; while a perforating lesion may give scarcely any indication of its existence until within a few hours of a fatal ending.

Such manifestations as diarrhœa, vomiting, pain, tenderness, and pyrexia are as much dependent on a co-existent enteritis or new growth as they are upon the ulcer. Doubtless the exposed surface of an ulcer offers the opportunity for irritation resulting in increased peristalsis, but this is not of necessity, for constipation may be present. Where the area of ulceration is extensive, the absorbing surface is by so much diminished, and thus while the general nutrition suffers, the unabsorbed products of digestion are liable to decompose and irritate.

Diagnosis.—From what has been said, the diagnosis of ulceration in this section of the bowel is frequently impossible, and an ulcer is assumed rather than proved to exist. In enteric fever, ulceration is taken for granted, though no special symptoms may indicate its presence. But if a severe and persistent diarrhœa, with liquid evacuations containing perhaps blood and shreds of mucus, and much pain and tenderness over the abdomen, supervene in a case of pulmonary tuberculosis, it is a fair inference to assume ulceration of the intestines, a diagnosis that would be confirmed by the discovery of the specific bacillus in the stools.

Prognosis.—This largely depends upon the cause. Except through perforation or fatal hæmorrhage by erosion of vessels, death does not take place from the ulceration itself. But a tuberculous ulcer is not to be expected to heal, and it may by its development hasten the end of a phthisical patient. The prognosis of typhoid ulceration will almost entirely be founded on the general state of the patient, since the indications of the bowel lesion may be so slight.

Since ulcers in this situation are inaccessible to direct **treatment**, little can be done for them apart from the general conditions which they may complicate, or the treatment of the symptoms to which they may give rise.

Recognising that an ulcer, when it exists, may lead to perforation, the object will be to avoid all undue movements of the intestines, and hence aperients are mostly forbidden, and opiates are indicated. Intestinal antiseptics, such as salicylate of bismuth or salol, are often of benefit by diminishing the putrefactive decompositions which are likely to exist. Rest is probably the only element of treatment that can affect the ulcerative process directly; while any improvement of the general condition should favour the healing.

Ulceration of the Colon

ULCERATIVE COLITIS—FOLLICULAR ULCERATION

The more characteristic forms of ulceration met with in the colon are described as regards their morbid anatomy and associations on pp. 166, 167, 174. Varying as the process does in extent from the single or several ulcers of a catarrhal, stercoral, thrombotic or hæmorrhagic, or neurotrophic origin to the widespread tracts of destruction seen in the follicular variety, and still more in the form specially designated "ulcerative colitis," the clinical characters exhibit a corresponding range in severity, from the simpler conditions, which may be difficult to distinguish from a catarrhal or membranous colitis, to the serious and fatal course exhibited by the graver lesions.

Whatever differences may exist in the causation of colon ulceration or in the precise anatomical features of the process, the **symptoms** are in their essential features very similar and vary chiefly in their intensity and duration. Of these *diarrhœa* and *pain* are the most marked and the most constant. The latter is noticed from the commencement of the illness, and is more or less constant, with frequent exacerbations, especially in connection with the action of the bowels, although tenesmus, such as characterises true dysentery, is seldom complained of. It manifests all degrees of severity, but is usually considerable and of a griping character, seriously adding to the progressive impairment of health which the patient exhibits. It is felt generally over the abdomen, and sometimes in the back and loins. There may be but very little tenderness, and such as there is is felt on pressure along the course of the colon. It is not easy to account for the pain, since extensive ulceration of the bowel may exist, as in typhoid fever or sometimes in tuberculosis, with little or no pain. It may be that the extensive denudation of the muscular

coat which takes place in severe ulcerative colitis may lead to the production of painful spasms, though this cannot be accepted as an altogether satisfactory explanation. The diarrhœa, with occasional intervals of constipation, is often most distressing, and further tends to the exhaustion of the patient. Ten or a dozen motions may be passed in twenty-four hours, consisting of dark fluid material smelling very badly, and usually containing some blood, fluid or in clots. There is, however, no great amount of mucus as a rule, although small clear discrete particles, and sometimes pus, characterise the stools of follicular ulceration. Sloughs are frequently passed.

Other symptoms are very variable and uncertain. Sometimes the abdomen is distended, and as the patient becomes thinner the movements of the intestine may become apparent. Nausea, vomiting, thirst, profuse sweating, and a furred tongue may all be met with, but are not constant. Nor is the pyrexia a regular phenomenon, although the temperature is usually somewhat raised, sometimes to 102° F. or higher; it is occasionally subnormal. The general condition of the patient slowly but steadily deteriorates; the anæmia, wasting, and debility progressively increase, in many cases to terminate in the "typhoid state" and in death, which is immediately due to exhaustion, or it may be to hæmorrhage or to perforation of the bowel and peritonitis. The coincidence of granular kidneys, which, as previously mentioned, is not infrequent, should be remembered in taking account of the general symptoms of this malady. Ulcerative colitis almost invariably tends to a fatal issue and may not last beyond a few weeks. Sometimes, however, it may continue for some months, with periods of temporary subsidence of the symptoms. In the follicular form of ulceration of the colon, death rarely takes place from perforation or from the bowel condition, but is due to some disease with which it is complicated, such as tuberculosis, although chronic Bright's disease is the commonest complication of simple ulcerative colitis (Hale White, *Guy's Hosp. Reports*, 1888). Infective pylephlebitis, hepatic abscess, and malignant endocarditis have each been met with as rare occurrences in this affection.

The **diagnosis** of an ulceration of the colon offers, as a rule, no great difficulty in the making, having regard to the symptoms, especially the pain, diarrhœa, and character of the stools, but the particular form of ulceration and extent of bowel involved is not always so easy to determine. Nor is this likely to be otherwise until the precise etiology of the several clinical forms of inflammation and ulceration of the large bowel, sometimes comprehensively designated "dysentery," be known. In acuteness and severity of

symptoms acute tropical dysentery stands first, and next probably the disease known as ulcerative colitis, then the follicular variety, whilst tuberculous and syphilitic ulceration and chronic dysentery have a more prolonged duration. A frequent examination of the stools is imperative to ascertain the nature and the progress of the case. Their appearance, odour, frequency, and the accompanying tenesmus in acute dysentery are characteristic, but in the more chronic forms they are less distinctive; the writer, however, agrees with Dr. Hale White in his opinion concerning the evacuations in ulcerative colitis that "so far as our present knowledge goes they never resemble ordinary dysenteric or choleraic stools; indeed they are so unlike them that probably the distinction is of considerable diagnostic value." A complete rectal examination should always be made, as the ulcers may be seen or felt, and the condition of the anus should be noted, as this is always involved in syphilitic disease of these parts. It is sometimes helpful also in distinguishing the colon ulceration from malignant disease when this is low down, and the settlement of this point is often difficult, as it is also in respect to chronic intussusception. The history of the onset and course of the case up to coming under observation is very important in these circumstances; and the presence of a distinct abdominal tumour is of still greater assistance. Should tubercle be suspected its presence must be sought for in the chest and elsewhere.

The **treatment** to be pursued in these cases may be summed up as a more thorough application of that laid down for colitis (p. 291). The success that has followed lumbar colotomy suggests its proposal in this serious disease, unless the general condition of the patient be hopeless.

Though the cæcum may be implicated in the ulcerative processes, and may be involved with the small intestine in typhoid or tuberculous affections, it is sometimes the sole part of the canal which is ulcerated. Excluding malignant disease and actinomycosis, which are in another category, ulcers in the cæcum are usually stercoral in origin, and their existence, unless perforation occur, with consequent perityphlitis, is seldom to be proved. The symptoms are those of typhlitis, already described (p. 290).

The appendix is a common situation for ulceration to be set up, though it would seem to be seldom involved in the more extensive affections, such as ulcerative colitis, follicular ulceration, or true dysentery. Any catarrh of its mucosa, whether simple or infective, such as typhoid or tuberculous, is liable to proceed to ulceration, but especially that which is associated with constipation and fæcal

accumulation, and when long continued leads to the formation of concretions composed chiefly of lime salts derived from the secretions, with a small proportion of fæcal matter; occasionally small hard masses of fæces may be found, and still more rarely (3 per cent, Treves) foreign bodies that have been swallowed, as seeds, shot, pins, etc. A superficial resemblance to cherry- or date-stones is sometimes to be noticed in the true concretions. The irritation set up by these objects on an inflamed mucous membrane readily induces ulceration, and such a lesion sooner or later extends through the thickness of the appendix, causing perforation and perityphlitis. Until this accident occurs it is doubtful how far any symptoms which may be present are distinctly referable to the appendix, any more than is the case in a simple catarrhal appendicitis. Pain and tenderness over the cæcal region, more precisely, perhaps, at the so-called "MacBurney's point," with possibly vomiting and a rise of temperature, may indicate the lesion, but it is very uncertain whether these or any indications are to be met with until the peritoneum is involved (perityphlitis), although no actual perforation may have occurred. (p. 347).

MALIGNANT DISEASE OF THE GASTRO-INTESTINAL TRACT

Cancer of the Stomach

The onset of the **symptoms** characteristic of this disease, the morbid anatomy of which is described on p. 180, is, like that of chronic gastric affections generally, insidious, and their exact commencement can rarely be indicated. In the cases occurring in early life the observations of Drs. Osler and M'Crae go to show a more abrupt commencement and more rapid course. In a small proportion of cases there is a history of dyspepsia for many years, and sometimes of very definite gastric ulcer, but far more frequently the patient gives no such account of his previous health.

The symptoms are those of a progressive indigestion, exhibiting much variation in the degree to which they are severally present, and it is from a combination of signs and symptoms rather than from any absolutely pathognomonic phenomenon that the diagnosis is inferred. In an appreciable number of cases the symptoms throughout, or until almost the end, are so slight that the real nature of the affection is unrecognised.

Among the earliest evidences of gastric mischief is *loss of appetite*

which is frequently extreme, now and then exhibiting perhaps a transient improvement. It is usual for this anorexia to have existed for some time before the patient comes under observation, which event commonly follows the onset of some more serious symptom. Cases, however, do occur in which no impairment of appetite has been complained of. The distaste for food may amount to actual nausea, especially at the sight of meals. Gaseous eructations, chiefly of CO_2 derived from the fermentations taking place in the stomach, are sometimes most troublesome, and to the same cause is due flatulent distension of the stomach.

Vomiting is one of the most constant signs, being rarely absent when the pylorus is the site of the growth, being less frequent when the cardiac end of the organ is involved, and may be entirely absent when the whole viscus is infiltrated with a scirrhous formation. The relation of its occurrence to the ingestion of food is variable, often not until an hour or two afterwards, or may be at once when the mucous membrane is very irritable. Whilst generally persistent throughout the course of the malady, it is nevertheless subject to variation in severity; and in the last stages, when there is much dilatation, it may almost entirely cease.

The vomit contains the remains of very partially digested food, some of which may have been swallowed days or even weeks previously, and with more or less blood, dependent on the extent of the ulceration of the growth. The blood is usually altered in character, and of "coffee ground" appearance; but an extensive hæmatemesis of bright blood may occur, though far less frequently than is the case with gastric ulcer. On the other hand, the amount may be so small as to require the microscope for its detection. Should the growth be sloughing, then portions may be recognised in the vomit, which is also likely to contain sarcinæ, torulæ, and other organisms associated with delayed gastric digestion, and with lactic acid and other fermentations. The odour of the vomit is occasionally most offensive, even where communication with the colon has not been established.

A valuable character of the contents of the stomach is to be found in connection with the hydrochloric acid of the gastric juice. The method of examination has been described on p. 269, and it has been observed in a very considerable number of cases that this acid is absent in cases of carcinoma of the stomach. So frequent is this that it becomes a sign of diagnostic value, inasmuch as no such constant absence occurs in gastric ulcer, with which cancer may be confused. Whilst repeated absence is strong evidence in

favour of cancer, it cannot be said that the presence of the acid in the material withdrawn from the stomach under proper precautions excludes the existence of this disease. In the absence of hydrochloric acid, lactic acid is commonly found, but sometimes both are wanting.

Even more constant than vomiting is *pain*, although cases have been known in which it has been, if not quite wanting, at least very slight; as a rule, it is severe and constant, of a dull, gnawing character, diffused generally over the gastric region, sometimes increased after food is taken, and very occasionally eased by the same, and seldom much relieved by vomiting. Some tenderness, perhaps extreme, is elicited on pressure over the epigastrium or gastric area generally, but there is seldom any specially localised point. Referred pains and tender points over the regions of the trunk and head are complained of; but "cutaneous tenderness is of less value in gastric carcinoma than in other diseases of the stomach, for cases of malignant disease are complicated by the presence of tender areas due to secondary deposits in other organs; moreover, in long-standing and exhausting diseases, pains often radiate far beyond the areas usually associated with the affected organ" (Dr. Hale White).

The *tongue* may be pale and moist, or furred, or dry and brown with red denuded edges. The *bowels* are variable, though generally constipated, and the motions may from time to time be black from sulphide of iron derived from blood. Indican is a very frequent constituent of the *urine;* otherwise no abnormality is to be found with any constancy, the diminished urea elimination being common to many wasting diseases with a small intake of food. Albuminuria and glycosuria may be present intermittently, and complete suppression may follow a copious vomiting; when also the gastric secretion is very considerable, the chlorides may temporarily disappear from the urine. An excess of creatinin has been noted.

The recognition of a *gastric tumour* is most uncertain. In many cases none is to be made out, though post-mortem examination may reveal one of considerable size. When situated at the cardiac orifice it is quite beyond detection, except by the passage of a stomach tube, and the same is sometimes the case with pyloric growths which may lie high up under the liver and margin of the thorax. Probably, on the whole, the mass is best to be felt when the stomach is empty. Tight lacing, by depressing the viscera, may make the tumour easier of detection. The situation of the pylorus, or of any growth connected with it, is liable to be displaced if there be much

dilatation of the organ, and especially if there be any proptosis; but, when palpable, a pyloric tumour is oftenest to be seen and felt in the epigastric or umbilical regions, rather more to the right of the median line, and much less frequently in one or other hypochondrium. A very imperfect notion of the real extent of the mass is obtained by manipulation, when it is palpable, owing to its connection with surrounding parts; it usually feels hard, and often nodular, giving a subtympanitic or dull note on percussion. As regards its mobility and the range of its movement with respiration the widest difference prevails, being quite fixed or freely movable, and shifting up and down with the movements of the diaphragm, or only perceptible on the deepest inspiration, or not even then. The physical signs of a dilated stomach usually coexist with those due to the tumour, and may indeed be the more prominent.

In those rarer cases where the entire stomach is implicated and the organ is much reduced in size with great thickening of the walls, or where the anterior wall is only or chiefly the seat of the growth, the tumour may be easily felt in the left hypochondrium, when it has been mistaken for an enlarged spleen, and with the characteristic alteration in the blood has led to a diagnosis of splenic leukæmia. The absence of any extension of the dulness upwards should serve to distinguish. The necessity for emptying the colon of scybala previous to examination is most important.

When the cardiac orifice is involved, the symptoms are those of œsophageal obstruction; there is dysphagia, and such food as is swallowed accumulates in great part in the gullet, whence it is regurgitated unaltered so far as gastric digestion is concerned.

General state.—The small amount of food taken, the serious interference with the digestion of that which is retained, and perhaps also the prejudicial effects of the metabolic products of the growth itself, combine to produce a great impairment of nutrition, which is intensified by the more or less constant loss of blood and the distressing pain. Hence a progressive *emaciation*, which commences quite early in the illness, and great muscular weakness are characteristic of the disease. Few conditions lead to so extreme a wasting, should the patient live long enough. A profound *anæmia* is soon established and contributes to the intense debility, and explains the œdema of the feet and ankles usually met with, and also the thrombosis of the veins of the limbs occasionally seen. In advanced cases, especially those of the medullary form with frequent hæmorrhage, the characters of the blood resemble those of the pernicious type of anæmia, though the diminution in number of

red corpuscles is seldom, if ever, so great; an apparent excess of red corpuscles has been seen when the fluid of the blood has been drained by excessive vomiting or hypersecretion in the stomach; some leucocytosis is to be observed, but nothing distinctive in the presence or absence of any particular form of white cell, although megaloblasts are wanting. In patients carefully observed and kept under uniform conditions a *pyrexia* reaching to 101° or rather higher is generally to be noticed. The cause of this is not satisfactorily known, but is generally ascribed to some toxin formed by the growth or produced in the course of the associated gastritis or local peritonitis. Shivering, with a rise of temperature to 104°, happens in a few cases. The general condition of ill-health, as represented by these symptoms, constitutes what is known as the malignant *cachexia*, the patient presenting a more or less characteristic appearance, of which the pallid yellowish integument is one of the most striking features. Patches of brown pigmentation on the face and extremities are also frequently observed.

The course of the disease is invariably towards a fatal termination, usually within a year from the time that the symptoms are declared, and not seldom within three or four months when occurring in the young. The scirrhous forms are somewhat slower in progress, and death may not take place for two years, or even rather more, whilst surgical interference claims in suitable cases still further to prolong life. Transient periods of slight apparent improvement, and even a slight gain in weight, after coming under treatment, are not infrequent. The patient usually succumbs to asthenia, hastened perhaps by a severe hæmorrhage or by some other complication. Within a few days or hours of death a persistent hiccough is apt to set in, and the patient lapses into a lethargic state which deepens into coma and may be marked by a mild delirium; unable to take any nourishment beyond an occasional teaspoonful of liquid, and too feeble to vomit, life gradually slips away.

The progress of the case may be complicated by perforation of the growth into the peritoneal or pleural cavities, or into some part of the intestinal canal, most frequently the transverse colon, when the vomit becomes fæcal and a diarrhœa is likely to be set up. The pressure of the growth itself or of secondary nodules in the liver or lymphatic glands upon the portal vein and common bile duct may lead to ascites and jaundice; and its extension to the liver and even the head of the pancreas, or the occurrence of secondary deposits in the liver or lungs, will give rise to special symptoms. The obstruction, both structural and functional, at the

pyloric orifice tends to induce some gastroectasis; and this will be greater in scirrhous stricture, though seldom so great as is caused by a non-malignant constriction. A rare complication is an umbilical fistula from adhesion of the growth to the abdominal wall. This lesion is much more frequent in tuberculous peritonitis or malignant disease of the omentum. The occurrence of nodules around the navel has been already referred to.

In a large number of cases the **diagnosis**, certainly at first, is conjectural, in the absence, that is, of an epigastric tumour being detected. In a person over middle life in whom dyspeptic symptoms such as anorexia, nausea, constipation, with discomfort at the pit of the stomach, and a growing weakness and anæmia, are developing, the suspicion of gastric cancer should arise; and the significance of such symptoms would be greater if the patient have previously enjoyed good health. Should there be actual pain and vomiting, the diagnosis will be still more probable, and the presence of blood with the absence of hydrochloric acid in the vomit will make it all but certain, the recognition of an epigastric tumour affording conclusive evidence. Enlarged supraclavicular glands and nodules around the umbilicus are strongly suggestive.

The conditions from which cancer of the stomach have more particularly to be differentiated are gastric ulcer, non-malignant pyloric growth or stricture, and pernicious anæmia.

As a rule, the pain of ulcer is more localised, more severe, directly follows the taking of food, and is relieved by vomiting, although exceptions occur to each of these statements. In cancer the pain is diffuse, more constant, and is less influenced by the ingestion of food or its ejection. The emaciation in the former condition is also far less marked, and gastric ulcer may even coexist with a very fair bodily condition. The presence or absence of hydrochloric acid from the stomach contents is generally reliable to discriminate between these two conditions, though this may not be so in the early stage of cancer, when the other symptoms are also uncertain. The age of the patient is an important element, and also the duration of the symptoms, especially the hæmatemesis; and it should not be forgotten that a chronic ulcer may be associated with sufficient inflammatory thickening to give rise to a very distinct tumour.

In distinguishing between a malignant and non-malignant growth of the pylorus, attention should be paid to the extent of gastric dilatation, to the degree of wasting, the chemical character of the vomit, and the presence of cachexia. Important as the

diagnosis in this case is with a view to operative treatment, it is often most difficult, and it is on the observer's unformulated experience that the final judgment often comes to be based.

The general similarity of the characters of the blood to those of pernicious anæmia renders the diagnosis of this malady almost excusable in those cases where the gastric symptoms, such as vomiting and pain, are in abeyance. Several cases have been recorded where the mistake has been made, and with some justification. The degree of leucocytosis in gastric cancer being but slight, should serve to distinguish from leucocythæmia, even though the contracted, thickened stomach should simulate a splenic tumour.

Some cases of chronic gastritis in persons in middle life closely resemble malignant disease of the stomach, and the not infrequent development of cancer in an organ that has been the seat of long-standing catarrh makes the diagnosis more difficult, especially when no tumour is to be recognised. A similar difficulty has occurred in distinguishing between cancer of the stomach or duodenum and lead poisoning. In all these cases, attention should be directed to the previous history and duration of the malady, the nature and seat of the pain and its relation to food, the occurrence of vomiting and the characters of the vomited material, the examination of the blood, and the existence of a cachexia.

The signs and symptoms of primary duodenal cancer closely resemble those of the similar gastric affection, and a differential diagnosis is often impossible. The situation of the pain and tenderness, and of the epigastric tumour when palpable, are perhaps rather more distinctly to the right of the middle line than when the growth is strictly pyloric, but this is not always reliable. Inasmuch as the obstruction caused by the neoplasm tends to cause gastrectasis, symptoms indicative of stomach disturbance soon occur. Jaundice from obstruction of the orifice of the bile duct is likely to supervene according to the exact situation of the growth.

Treatment.—Although so far operative procedure can only claim success in exceptional cases, it gives promise of improved results; but this very much depends on an earlier recognition of the disease than at present is usually the case, and an exploratory laparotomy, now comparatively free from risk, is to be reckoned as one of the means of ascertainment. Complete resection of the stomach, and excision of the pylorus, have each been performed with success and considerable prolongation of life, and even complete freedom from suffering, but the cases have been extremely few. Somewhat more promising are the records of gastro-enterostomy

when the disease has been confined to the pylorus; and a gastrostomy affords most relief and a short extension of life for cancer of the cardiac end. But the postponement of operation until a definite tumour is to be felt greatly diminishes the chance of benefit, since by that time extension of the disease to adjacent structures or secondary deposits is likely to exist. It may, however, be observed that operation is not infrequently followed by prolonged improvement in health, even in cases apparently *in extremis*, possibly by diverting the food and so diminishing the irritation of the ulcerated surface.

Since no other means of cure are possible, all that remains is to prolong life as far as is possible by the relief of symptoms, and this may be done in some measure by attention to diet and with the help of drugs. Having regard to the impaired digestion that takes place in a stomach which is the seat of cancer, when the efficiency of the gastric juice is diminished by lack of acid, and probably with pyloric obstruction and dilatation of the organ, and in any case impaired motility, it is clear that the gastric contents are very apt to become, from abnormal decompositions, very unhealthy, and to that extent a source of irritation. Hence washing out the organ is distinctly indicated, and when this can be regularly performed every day or every other day it affords the greatest relief to the patient even from the pain and sickness, and gives the best chance for the digestion of such food as is taken. The difficulties, however, are often great in pursuing this course, owing to the insuperable objections of the individual; for although no doubt lavage causes little or no discomfort when once the habit is acquired, it is distinctly unpleasant in its first practice to most people, and this especially so when begun on a patient who is seriously ill with malignant disease of the stomach, and promises of benefit will often fail to allow a fair trial of the method. If submitted to, it may be said that not even hæmorrhage contra-indicates its employment. The chief points to observe in respect to the food are that it should be given in small quantities at intervals of three or four hours: a little digested and passed on into the intestines is better than larger amounts which decompose and irritate; the bad appetite will generally prevent any overloading of the stomach. Considerable latitude may be given to the patient's own wishes as to what he takes, but it is well to avoid, as far as possible, fats and much sugar or starch food, as well as vegetables, and the more nitrogenous the diet is the better. Fluid food also should only be given in small quantity. Minced meat, chicken or fish, and eggs are all

permissible so long as they can be taken. Peptonised foods, including milk, Benger's food, plasmon, soups, and meat extracts come to be the nutriment that has to be relied on, and these may have to be supplemented by nutrient enemata or suppositories. Alcohol in the form of sherry, whisky, or brandy, or champagne iced, especially if there be much sickness, may be given in amounts proportioned to the habits of the patient and his condition. Fruit jellies or the juice of fresh fruits, and grapes, are often grateful to the sufferer. The appetite may be improved and the digestion aided by giving full doses of hydrochloric acid with vegetable bitters, gentian, quassia, chiretta, or condurango, and pepsin may also be added for the same purpose. Distinct benefit follows this treatment.

Antiseptics to check fermentations in the stomach are only of moderate advantage; creasote, carbolic acid, resorcin, and many others are used, but salicylic acid is one of the best.

Sooner or later morphia has to be administered by mouth or hypodermically for the relief of the pain, but previous trial should be made of hydrocyanic acid in an effervescing form with bismuth, which is conveniently given in the form of liquor bismuthi et ammoniæ citratis; codeia; or of potassium iodide with bismuth, which sometimes gives distinct relief. Aqua chloroformi will often answer the same purpose.

The vomiting, as already said, is best treated by lavage, otherwise the effervescing hydrocyanic acid and bismuth mixture, with or without three drops of liq. morphinæ acetatis or of liq. cocain. hydrochlor., or iced champagne may be tried. It is sometimes well to encourage the vomiting by swallowing a tumblerful of warm water, which washes out the stomach; but the efficacy of many of these remedies will depend on whether the organ be dilated and whether it contain a quantity of ill-digested material.

The constipation, which is often very troublesome, may be met by laxatives such as cascara sagrada, or by simple enemata or glycerine suppositories.

Cancer of the Bowel

The anatomical characters and the usual situations for the development of malignant new growths of the intestines are described on pp. 183, 184.

Symptoms.—For a varying time before this disease definitely asserts itself, the patient may complain of vague dyspeptic symptoms; loss of appetite; a sense of uneasiness in the abdomen, not amounting

to pain, and usually increased after meals; and marked irregularity in the action of the bowels, with or without flatulent distension. There is an increasing tendency to constipation, for which the patient often takes large quantities of aperients of a drastic and cathartic character, which one by one lose their effect. The persistence and gradual increase of these symptoms, especially if there be any loss of flesh, are very significant, and should excite suspicion. Sooner or later, according to the duration of the case, the usual cachexia is established; and in the greater number of cases the patient rapidly emaciates, especially towards the end, though in cases of very short duration the wasting may not be so excessive. The emaciation depends not only on the general perversion of nutrition caused by the development of the cancer, but also on the direct influence it exerts on organs concerned in the digestion and absorption of nutriment; hence the higher up in the bowel the growth is situated the sooner is the loss of flesh determined.

The local signs and symptoms referable to the new growth itself are very variable in their occurrence, and often are singularly slight in comparison with the gravity of the cause. Thus *pain* may be completely wanting, and perhaps there is but little tenderness on pressure; when present the pain is usually of a dull character, although it may attain a severe degree, and be quite localised—sometimes specially referred to the loins and passing down the thighs.

The indications of the tumour produced by the new growth are very uncertain, being often little more than an ill-defined fulness in one region, scarcely even amounting to that in the annular form of cancerous stricture; at other times presenting a distinct hard irregular mass of variable size. This last quality, being partly dependent on fæces, is very characteristic. Should the growth happen to be situated over the aorta or iliac arteries, an indistinct pulsation may be communicated to it. The percussion note over the tumour is usually imperfectly tympanitic, from the existence of coils of intestines between it and the abdominal wall, the thickness of which will of necessity considerably modify the signs of the existence of the growth. The mass may present all degrees from free mobility to complete fixity, dependent on the nature of its seat and also on the existence of adhesions to eighbouring parts. By pressure on veins or on nerves, œdema and pain of the lower extremities may be caused.

Symptoms of *intestinal obstruction* are rarely wanting, and may be the first for which the patient seeks relief. Vomiting, constipation of increasing severity, with signs of intestinal distension above the lesion,

are among the most constant. The character of the vomit will largely depend on the duration and situation of the lesion, and the actual degree of obstruction. When the jejunum or lower part of the duodenum is the seat of the growth the vomiting is more copious and constantly bile-stained, the bowel and stomach above the stricture sharing in the distension. When the obstruction is lower down, in the large intestine, the vomiting may be slight, and consist of little more than the gastric secretions, with such food as is taken, only becoming stercoraceous when the obstruction becomes complete. Occasional diarrhœa, determined by the chronic enteritis which exists, may alternate with the constipation; and rupture of the intestines, especially the cæcum, has been met with as a result of the atrophy and ulceration of the over-distended, chronically inflamed bowel. A moderate degree of pyrexia is not infrequent, due probably to septic absorption from the ulcerated growth, or to an associated enteritis. As in other forms of intestinal obstruction, the amount of urine voided is likely to be very much diminished when the stricture is high up and the vomiting is excessive (see Intestinal Obstruction, p. 250). The *stools* may be characteristic of the obstruction, consisting of small separate masses, frequently hard and round, or flattened or in thin cylinders ("pipe-stem") if the growth be near the anus; but perfectly normal-shaped motions may be passed even with a stricture as low as the sigmoid flexure (see p. 254). They may be mixed with sloughed-off portions of the new growth, or with blood that has escaped from the ulcerated surface. If the peritoneum be involved peritonitis may arise; and ascites, often considerable, is usually developed with colloid cancer.

Superadded to these symptoms will be those caused by the morbid condition of any other organ that may be affected, such as the liver, bladder, or uterus.

Primary carcinoma of the appendix (of which Dr. Rolleston has collected a series of cases, *Lancet*, 7th July 1900), when it gives rise to any symptoms, may simulate those of appendicitis.

Course and terminations.—Malignant disease of the intestines in the majority of cases progresses continuously. It is difficult to state even an average duration, owing to the insidious onset and vagueness of the first symptoms; but the greater number of cases do not go beyond twelve to eighteen months from the time when the disease is clearly established, while some may be fatal in a few weeks, and a few may last for several years.

Death may result as the direct consequence of the cachexia;

or from complete intestinal obstruction, hæmorrhage, peritonitis, or other effects of the growth.

Diagnosis.—An exact diagnosis is often not to be made, and the nature of the case remains throughout uncertain, if not actually as to the existence of a malignant growth, at least as to the seat of it. The earlier insidious and ill-defined symptoms present nothing diagnostic, though their progressive character and resistance to treatment would cause a suspicion, especially in a person over middle age, and in whom a gradual, even though slight, loss of weight is noticed. Even in the later stages the symptoms are almost identical with those of chronic enteritis, which co-exists with the new growth to a greater or less extent; and in the not infrequent cases in which a tumour is not to be felt, or is uncertain in its indications, the diagnosis becomes extremely difficult.

Supposing that the existence of an abdominal tumour be clearly ascertained, it is not always easy to determine its connection with the intestine, since the variability in position, in mobility, and in size (due to the accumulation, or the reverse, of fæces) precludes any diagnostic sign, although this very variability is regarded by some as almost indicative of intestinal cancer. In distinguishing between an intestinal tumour and one connected with the liver, gall-bladder, pancreas, kidney, mesenteric glands, uterus, abdominal wall, or the inflammatory thickening following a perityphlitis, an aneurysm, or a simple fæcal accumulation, the history of the case, age, progressive nature of the condition, existence of tumour, signs of obstruction, and character of the stools, are the points to be considered in forming a diagnosis. Any one or even two of these might equally indicate other lesions, but, taken collectively, they will usually justify the formation of an opinion. A rectal cancer, which is accessible to the touch or even to inspection, need offer no difficulty, but it is otherwise where it is a question of distinguishing a duodenal growth from one strictly limited to the pylorus. A firm epigastric tumour, felt close to the margin of the thorax, and associated with distinct dyspeptic symptoms, may also be due to primary cancer of the head of the pancreas; in such case the symptoms of obstruction are less marked at first, but the growth will probably involve the bowel in its progress and cause stenosis; owing to the destruction of the gland tissue the pancreatic juice is not secreted, and undigested fat may be found in the stools. Similar difficulties may surround the investigation of a tumour situated in the right iliac fossa. The importance of completely emptying the bowels by enemata, before finally forming a diagnosis,

cannot be too emphatically insisted on, since the signs, and in many respects the symptoms, of intestinal cancer may be simulated by a fæcal accumulation due to simple atony of the colon. The anæsthetisation of the patient is also frequently necessary before the exact situation, or indeed the existence, of a growth can be determined.

Treatment.—The extremely rare cases of reputed natural cure of malignant disease of the intestines, brought about by sloughing of the growth and subsequent cicatrisation of its site, afford no hope of our being able to imitate the process artificially, and the medical treatment remains at the best symptomatic and palliative.

The diet should be so arranged as to contain the minimum of indigestible residue, and permit the chief digestion and absorption to take place in the stomach, if it be the upper part of the tube that is affected. But in the majority of such cases the utmost disinclination for food exists, even apart from any vomiting or pain which its ingestion may produce, and hence, whatever the directions, the patient in the later stages practically takes nothing. The anorexia is frequently as marked even when the mischief is seated in the colon, and the area for digestion and absorption is uninterfered with.

In the earlier stages it may be advisable to insist upon as much nutritious food as possible being taken, either by the mouth or in the form of enema or suppository, so as to offer the most prolonged resistance to the inevitable end; but at the same time there is no slight reason for thinking that the same course favours the development of the new growth. Preparations of iron may be given.

The symptoms dependent upon the obstruction are open to little relief short of operation. Only the mildest aperients are permissible to combat the constipation, and these should be salines with the object of rendering the fæces as fluid as possible; while the vomiting is as a rule uncontrollable, and, indeed, is often a relief.

Hæmorrhage may require special attention, the tincture of hamamelis being one of the most effective styptics. The pain may be so severe as to necessitate free administration of morphine subcutaneously; in other cases belladonna is of value in alleviating the local discomfort, and acts favourably by allaying any spasm.

DILATATION OF THE STOMACH AND BOWELS

Dilatation of the Stomach—Gastroectasis

The **signs** and **symptoms** of this far from uncommon morbid state exhibit a considerable variety in degree, dependent on the extent to which the distension of the viscus has taken place, as well as in some measure on the primary causal condition, the specific manifestations of which will complicate those of the gastroectasis. The causes of the dilatation naturally group themselves into those of an obstructive and those of a non-obstructive character (see p. 189).

Physical signs.—Some bulging of the abdomen over the dilated organ is generally perceptible, especially if the patient be at all emaciated. When the distension is considerable the line of the upper curvature may be seen in the epigastric region, with a depression just below the ensiform cartilage. The outline of the greater curvature is not always visible, but sometimes almost the entire contour of the organ may be apparent. When the condition is secondary to pyloric obstruction, and there is hypertrophy of the muscular coat, exaggerated peristaltic movements may be seen spreading downwards over the organ from left to right, and the stomach may at the same time be felt to harden under the hand applied over it. These movements occur at frequent and irregular intervals, and may be excited by gentle irritation of the surface; they serve to indicate the limits of the distended viscus, and are most apparent when it contains food or gas.

The lower margin of the dilated stomach may be felt on careful palpation, unless the parietes be unduly thick, but this is a sign that is on the whole less certain than are the other results of physical examination.

Far more reliable is the evidence to be obtained by percussion, for by that means the outline of the stomach may generally be satisfactorily ascertained. Allowance must be made for the presence of food or liquid in the organ. The characteristic "stomach note" may be obtainable upwards into the axilla as high even as the fourth rib, and in that situation it is at once detected. The lower limit, however, may be very difficult to distinguish from the colon, which lies just below, but Sir William Broadbent has pointed out that the distinction may be made by auscultation, "the cardiac sounds, especially the second, being heard all over the area of resonance

proper to the stomach, with a characteristic tympanitic consonance, which is lost the moment the stethoscope passes from the stomach to the colon."

One of the most distinctive signs of gastroectasis is a splashing sound audible on disturbance of the stomach contents, as by sudden pressure on the gastric region, or by shaking the abdomen; from time to time the sound is elicited in the course of the peristaltic movements of the organ.

The physical examination of the patient should be made both in the upright and recumbent position, and the several facts may be more precisely ascertained by the passing of an œsophageal bougie, and the position of the tip recognised by palpation through the abdominal wall; or the extent of the distension may be better defined by inflation of the stomach by gas such as may be liberated by swallowing in successive portions the constituents of a seidlitz powder suitably dissolved (see pp. 256-261).

The severity of the **symptoms** is not always proportionate to the degree of dilatation as shown by the physical signs. A considerable distension may exist with but little symptomatic disturbance. It is convenient to distinguish the manifestations directly referable to the stomach lesion from those which depend on the perversion of digestion and consequent malnutrition.

Of the former, *vomiting* is the most significant and the most constant. It usually occurs at intervals of twenty-four hours and longer, and frequently at the latter part of the day. The amount brought up is considerable, amounting to several pints, and contains a large proportion of the food taken many hours previously. The material is extremely acid in reaction, and of sour odour, from the organic acids derived from the decomposition of the gastric contents; the bulk is largely increased by an excessive secretion from the glands. Sometimes the vomit is of a brownish colour, and it may contain altered blood as well as abundant micro-organisms, of which sarcinæ and torulæ are the most numerous. It may continue to ferment after being voided if kept in a warm place, and forms a yeasty scum on the surface. The ejection of the material is commonly very sudden and forcible, and unaccompanied by retching or much nausea.

Gastric *flatulence* and gaseous eructations, chiefly of CO_2 derived from the fermentations, are other marked symptoms. The enormous formation of gas increases the distension of the organ, and though fluctuating in its intensity, adds considerably to the distress which its condition entails (see p. 208).

The extent to which discomfort or *pain* in the stomach is complained of varies much, sometimes they are scarcely existent, and they bear but little relation to the taking of food. Rather is it that pain is felt before vomiting occurs, and is relieved by the emptying of the stomach.

Owing to the inadequate passage of food onwards into the intestine, whether from the vomiting, or the pyloric stenosis, or from both, the patient tends to deteriorate in general health, and to lose weight from want of sufficient nourishment, however much may be actually swallowed. For the same reason *constipation* is complained of, there not being enough bulk of material to excite intestinal peristalsis. The excessive drain on the blood by the hypersecretion determines a *thirst*, the relief of which only adds to the distension of the stomach. The *urine* is generally scanty and concentrated, and contains an abundance of ethereal sulphates derived from intestinal decomposition. Vomiting of large quantities of fluid may be followed for some hours by anuria. In some cases certain *cardiac symptoms*, especially palpitation, irregular action of the heart, and even pain of an anginous character and breathlessness, are very distressing. They are mainly due to slight displacement of the heart and interference with its action by the dilated stomach, and the relief of the distension by eructations of gas or by vomiting removes the cardiac distress.

The retention of the ingesta in the stomach and the abnormal fermentation resulting therefrom may reasonably be supposed to lead to the formation of substances which are irritant and toxic. A catarrh more or less persistent is established in the mucosa, and the tongue becomes coated and the breath foul.

Foremost among the symptoms which may be ascribed to the absorption of toxic substances from the stomach are *headache*, and *alterations in mental state* varying from irritability to depression, which may be extreme. With this go considerable physical prostration and disinclination for exertion, both muscular and mental. In the severest cases the *torpor* and *lethargy* deepen into coma, which may be fatal. *Cutaneous eruptions* of an erythematous or urticarial character have also been noted in the course of the malady, presumably toxæmic in origin. And to the same cause is perhaps to be assigned the *vertigo* which often occurs. A form of *insomnia* which sometimes arises is distinguished by the patient waking regularly after the first few hours' sleep, and remaining awake the rest of the night in spite of all ordinary efforts to sleep, and perhaps dropping off into a heavy slumber from exhaus-

tion when the night's rest should have terminated. This has been attributed to flatulence, and if this can be relieved by rubbing the abdomen, walking about, a draught of a hot carminative, etc., sleep can be induced. It cannot be said that this symptom is in any way peculiar to gastroectasis, although it is distinctly characteristic of some perversion of gastric digestion, and is very liable to occur in persons the subject of mental anxiety or strain in whom digestion is delayed. One of the most interesting symptoms associated with gastroectasis which the recent researches of Drs. Halliburton and M'Kendrick have gone far to show is toxic in origin, is the so-called *gastric tetany*. It is not of very common occurrence, and is marked by contraction of the fingers or toes, or both, the spasm sometimes involving the arms and legs, and more rarely the muscles of the trunk, neck, and face. Alternating with the tonic seizures may be clonic convulsions, though these are sometimes very slight. The attacks are often preceded by severe pain in the stomach, and are occasionally accompanied by unconsciousness. Washing out the stomach or vomiting frequently induces the paroxysm. The co-existence of transient albuminuria has been occasionally noted, but this is not to be regarded as indicating the cause of the tetany to be uræmic in nature, but rather is it the result of the renal epithelium being damaged by the elimination of the irritant toxin.

Acute dilatation.—In the rare cases of gastric dilatation of sudden onset, usually terminating fatally in a few days without any obvious cause, and apparently of the nature of an acute paralytic distension with enormous hypersecretion, the patient quickly passes into a very grave state of collapse. The physical signs of the condition exist in a marked degree, vomiting and gastric pain are manifested until the prostration is far advanced, and the emptying of the stomach is rapidly succeeded by a further copious secretion of fluid, which may be bile-stained; the skin is dry and shrunken, and the urine almost or quite suppressed.

Intermediate, as it were, between such cases as these and the more chronic forms just described, are those which complicate acute infective diseases in which the symptoms for the most part correspond to the description given, but are more quickly established, and perhaps sooner recovered from.

Diagnosis.—This should not be difficult, at least so far as the recognition of the existence of the dilatation. A careful consideration of the physical signs, of the character of the vomiting, and nature of the vomit, and of the remote symptoms, should be

sufficient to establish the diagnosis, though it may not always be easy to exclude extreme dilatation of the transverse colon. But it is otherwise as regards a diagnosis of the cause of the condition, and for that other circumstances have to be considered, such as the history, presence of an epigastric tumour, character of the vomit in respect to the amount of hydrochloric acid, the presence of blood or of bile, the frequent appearance of the last suggesting that the causal obstruction is in the lower part of the duodenum. Some assistance is obtained by remembering that the cases of greatest distension are often due to non-malignant stenosis of the pylorus, and that cases of non-obstructive causation give physical evidence of more distension upwards, the stomach on the whole tending less to fall in the abdomen.

Prognosis.—Reference has been made to the usual fatality attending the very acute cases, and those of more chronic character are mainly influenced in their course and termination by the cause, death being due in malignant obstruction to other circumstances. Among the symptoms of serious import tetany is prominent, but even this may be diminished in its gravity by prompt treatment. Besides being directly responsible for a fatal termination, gastrectasis is not infrequently the immediate cause of death in persons the subject of cardiac degeneration, in whom the disturbance of the heart's action by the dilated stomach is, so to say, the "last straw."

Treatment. — Undoubtedly much may be done for this condition. Whilst cure is not possible, from the nature of the cause in some cases, relief is possible for almost all. The question of the treatment of an existing pyloric stricture is dealt with elsewhere, but for the dilatation itself, however it have originated, the main indications are to prevent accumulations in the stomach, and to check fermentation therein.

The vomiting is the natural effort to relieve the first indication, which is even more efficiently met by emptying the stomach with a tube at regular intervals, and subsequent washing out of the organ. This should be done once in the twenty-four hours, preferably in the evening, and the lavage should be carried out with several pints of water at a temperature of 85° Fahr., in which boric acid (4 drachms to the pint), or sodium bicarbonate in the same proportion, is dissolved. By this means the decomposing food stuffs are removed and their toxic effects are avoided, and at the same time the mucous membrane is given a chance of recovery. It is not, of course, claimed that such a treatment will cure the dilatation con-

sequent on a permanent pyloric stricture, but it goes far to do so when the underlying cause is atony and a chronic catarrh.

Carbolic acid or creasote in one or two minim doses, hyposulphite of soda, or sulphocarbolate of soda, grs. 5 to 15, made up with carminatives, are the most effective antifermentatives. The co-existing catarrh is much benefited by potassium bichromate in $\frac{1}{10}$ to $\frac{1}{8}$ gr. doses at meal time in a pill or capsule.

Much benefit in restoring the tone of the stomach may be derived from supplementing the lavage by abdominal massage, with or without applications of faradic electricity for five to fifteen minutes, of sufficient strength of current to cause distinct contractions of the abdominal muscles, one pole being placed on the stomach and the other round towards the back. The "toning up" of the gastric musculature is even still more effectively brought about by the administration of high frequency currents daily or on alternate days for ten to twenty times.

The diet and feeding of such cases are of great importance. Sugar and starch foods, as being liable to ferment in the stomach, should be reduced to a minimum. Minced freshly cooked meat, peptonised milk, Benger's food, and plasmon are the most suitable, and should be given in small quantities at intervals of four hours, preceded with an occasional dose of dilute hydrochloric acid. All liquids should be given sparingly. Rectal feeding may be necessary at first to give the stomach as complete a rest as possible, the lavage meanwhile being continued, and this mode of nourishment is the only one feasible in the cases of acute dilatation. When the condition is only moderate in degree a diet restricted to lean meat and hot water may be most beneficial.

When the primary cause is atony of the stomach, strychnine, arsenic, and iron, with general tonic treatment and good hygienic surroundings, are indicated.

The surgical treatment of the condition may consist of excision of the pylorus, and union of the duodenum to stomach (pylorectomy), which is resorted to in favourable cases of pyloric new growth; or in non-malignant stenosis the orifice may be forcibly stretched. The dilated organ has been frequently reduced in size by making folds on the anterior surface, from the lesion to the greater curvature (gastroplication). The operation has been most successful in its results when combined with pyloroplasty.

Dilatation of the Bowels

Abnormal distension of the intestines is of frequent occurrence, sometimes of the entire canal, but oftener of certain sections only, the colon being most commonly affected (see p. 190).

The **signs** and **symptoms** will vary with the extent of bowel involved and the degree of dilatation, but impaired peristalsis is the most constant result, apart from the enlargement of the abdomen, causing constipation or intensifying that which already exists, and that has contributed to the production of the distension. The swelling of the belly may be enormous, the integuments are tense and shiny, and a tympanitic note is to be obtained in percussion over the whole region. Should there be much emaciation the outline of the bowels, especially the colon, may be visible; and should the dilatation be dependent upon obstructive rather than simple atony or paralysis, the peristaltic movements in the coils of the small intestine may be perceived. Borborygmi, and sometimes splashing sounds, are to be recognised with and without manipulation of the abdomen. The pushing upwards of the liver and encroachment on the thorax, as well as the impediment offered to the movements of the diaphragm and expansion of the lungs and heart's action, lead to dyspnœa, palpitation, and cardiac distress, which may seriously imperil life.

Should the condition be at all chronic, symptoms of fæcal intoxication are likely to develop, prominent among them being mental depression and hypochondriasis (see p. 229).

The so-called "idiopathic" dilatation of the colon is often fatal; otherwise the distension is a complication that by the embarrassment to the breathing and to the heart may jeopardise life, and add to the gravity of the underlying cause.

It is often important to distinguish whether the small intestine or the colon be dilated, and where the condition is extreme this may be very difficult owing to the great size to which the ileum may be stretched. It may be possible to mark out a preponderance of distension in a particular region, and visible peristalsis indicates involvement of small rather than of large bowel.

The **treatment** should be directed towards the relief of the obstruction if this be possible, and stimulation of the musculature in the atonic cases, thereby preventing the accumulation of flatus, the formation of which may in a measure be controlled by antiseptics, of which salol is one of the best. If excessive the removal of the gas

may be a matter of urgency, and it is claimed that this may be accomplished by passing per anum a long tube; to the writer the effective possibility of this procedure is very doubtful. Stimulant enemata of turpentine or of cajuput oil are often useful if the distension be only moderate. But in extreme cases, when life is threatened by the distension, stronger measures are needful, such as tapping the gut with a fine trocar, or even opening the colon in the loin, a method which allows of subsequent local treatment of the bowel. Excision of an enormously dilated sigmoid, with restitution of continuity of the colon and rectum, has been performed successfully. Simply placing the patient in the knee-elbow position has sometimes been followed by great relief, and should always be tried. Massage and electricity as for gastroectasis should be carried out in the atonic cases, coupled with the administration of strychnine, exercise, and general hygienic treatment. Such articles of diet as furnish in their decomposition CO_2, H, SH_2, or CH_4, being chiefly those of a vegetable character, should be avoided as much as possible.

W. H. Allchin.

SPRUE

This is a peculiar form of chronic diarrhœa characterised by copious, liquid, white, gray, or straw-coloured frothy acid motions, limited in recent and typical cases to the early morning and forenoon, usually accompanied by denudation, more or less general, of the mucous membrane of the alimentary tract, and leading to atrophy of the muscular and mucous coats of the intestine; of frequent occurrence in tropical and sub-tropical countries, particularly in European residents, and never originating in temperate climates, except in persons who have lived in an endemic area.

"Diarrhœa alba" or "White Flux," in reference to the character of the stools, and "Tropical Diarrhœa" on account of its occurring in hot climates, are the names given to it by Indian writers. In the far east, where the denudation, especially of the tongue and mouth, is more constant and marked, it is called "Sprue" from a word used in the Scottish lowlands and in Holland (Spruw) for aphthous conditions of the mouth in children, which was first used by the Dutch in Java to designate this disease. *Psilosis linguæ vel mucosæ intestini*, the name proposed by Dr. Thin, from ψιλός, *bare*, also emphasises this condition of the mucous membranes.

Etiology.—This form of diarrhœa is most frequently met with in persons who have lived for a considerable time in a tropical climate. Long residence is not, however, essential, and cases are on record in which it has only been for a few weeks. The disease may be latent, and only develop many years after the patient has left the endemic area, and without his manifesting any symptoms in the interval. In India a large proportion of the cases originate in an attack of that closely allied, if not identical condition called "Hill Diarrhœa." Any exhausting illness, especially such as entails symptoms of intestinal irritation, as dysentery, a tendency to diarrhœa, fistula, and hæmorrhoids, predisposes to it, as well as uterine hæmorrhages, frequent child-bearing, and any depressing influence, such as syphilis, courses of mercury and iodide of potassium, bad food, etc. The sexes are equally liable to it, but it does not occur in children. Malaria cannot be cited as a cause.

Exhaustion of the vital forces is, however, insufficient alone to produce the disease; everything points to the probability of an invasion of the digestive canal by some organism, which may remain dormant for a considerable, sometimes a lengthened period, until conditions arise favourable to its growth and multiplication, when it finds in the altered conditions of the contents of the bowel a suitable medium, and its appropriate pabulum in the colouring matters of the stools.

Geographical Distribution. — It probably occurs in all tropical and many sub-tropical countries. It is particularly frequent in Java, the Straits Settlements, Ceylon, India, South China, Manila, Cochin China, tropical Africa, and the West Indies. It has also been reported in North China, and even in Japan. It never originates in temperate climates, except in persons who have resided at some time in some of the countries enumerated, but it may do so many years after their return to Europe.

Morbid anatomy. — The most characteristic pathological changes in this disease are found in the small intestine towards its lower end. The bowel is thin, attenuated, and diaphanous, and may be the seat of lardaceous degeneration. The serous coat is normal, the muscular atrophied, and the submucosa firm from abundance of fibrous tissue. The mucosa is profoundly altered. It is thin from interstitial atrophy, the villi are flattened out so as to be barely perceptible, and the glands have to a great extent disappeared. It is covered by a layer of dirty structureless mucoid substance, containing a few leucocytes and shreds of Lieberkühn's follicles, and on removing this mucoid layer, which gives a velvety

feeling to the touch, the surface of the attenuated membrane exhibits superficial ulcerations in the form of minute circular or serpiginous erosions or abrasions with pin-head pigmentation, and thin cicatricial patches of previous disease. Small cyst-like dilatations of the follicles containing muco-pus may be found.

There may be similar extensive disease throughout the large bowel, with ulceration, especially of the cæcum, sigmoid flexure, and rectum. These changes are most frequently found in cases which have followed on dysentery. The lymphatics in the neighbourhood of the ulcers may contain pus, and the mesenteric glands are often enlarged. The œsophagus in some cases has its epithelium and glandular structures destroyed with inflammatory exudation in its sub-mucosa and muscular coat. The stomach and duodenum usually escape, but a few small superficial circular ulcers are occasionally found in those parts.

The other thoracic and abdominal organs exhibit only changes attributable to general wasting. The liver is diminished in size, but there are no atrophic changes or cirrhotic contractions. The spleen is unaltered, but disease has sometimes, exceptionally, been found in the pancreas.

Stools.—The contents of the bowels, both large and small, are of a grayish white or faint drab colour, and acid in reaction. The acid reaction is due to the failure of the secretions of the diseased intestine to neutralise the acidity of the chyme. This acid reaction profoundly alters the character of the bacteriological flora of the intestinal contents. The bacillus coli communis is reduced in numbers, or may be almost entirely absent, and its place taken by swarms of micro-organisms, sometimes in nearly pure cultures. Different bacteria predominate in different cases, and no specific importance can yet be given to any.

The stools have been found to contain bile acids and fats, with an absence of hydrobilirubin, and to the destruction of the last the white colour may be largely attributed. There is no reason to think that the function of the liver is materially affected in this disease. If the patient vomit, which he rarely does, normal bile will be found in the vomited matters, and the white stools may persist for many months without jaundice or any symptoms referable to cholæmia or absence of biliary excretion. The urine does not contain bile as in biliary obstruction. The bile acids are sometimes in small quantity, but it must be remembered that the liver undergoes progressive diminution in size until the establishment of convalescence, when it regains its normal dimensions. The destruction

of the colouring matters of the stools is characteristic of this form of diarrhœa, and its cause is to be sought for in the abnormal bacterial conditions.

The presence of a large amount of fat in the stools has suggested functional inactivity of the pancreas, as contributing to the phenomena of sprue. Experiments have, however, shown that a very considerable proportion of fats ingested is digested and absorbed, but the extreme emaciation reached by the patient in this disease shows that this function is very imperfectly performed, and a large proportion escapes with the motions.

Active fermentative changes take place in the bowel contents, with the evolution of much gas, giving rise to much flatulent distension and frothy motions.

Symptoms.—The disease generally begins insidiously with morning bilious diarrhœa. This persists and soon changes to the pale liquid motions of the early morning hours, which distinguish this form of diarrhœa. The motions are then urgent, copious, watery, frothy, grayish white, and acid in reaction. They may be only one or two in number in the twenty-four hours. There is often much flatulent distension of the abdomen, which is relieved by the discharge of the contents of the bowels, after which the patient may in recent cases suffer little or no inconvenience and follow his usual avocations during the rest of the day. Gradually the diarrhœa becomes more frequent and occurs at other parts of the day, though even in the worst cases, with eight or ten motions a day, it never entirely loses its character of being a morning diarrhœa. Sooner or later the tongue assumes a peculiar raw-beef appearance and becomes very sensitive to condiments, wine, spirits, and hot or highly seasoned food. This is due to denudation of its epithelial covering, which may occur in patches and at the tip and edges before it becomes general. Superficial erosions and fissures, patches of herpetic eruptions, especially on the under surface, near the frenum, as well as on the buccal and palatal mucous membranes, may be seen. From similar conditions of the gullet swallowing is sometimes painful and difficult. The patient gradually becomes emaciated, haggard, and anæmic, the thinness and looseness of the skin of the limbs and chest, and the drawn expression of the face, being in marked contrast with the drum-like distension of the abdomen. The tongue ultimately becomes smooth, glazed, shiny, very red, or when the general anæmia is advanced, small, yellowish white, and creviced.

The progress of the symptoms, which may continue for many

months or several years, is not uniform. It is often interrupted by periods of comparative well-being of some days' or weeks' duration, when the diarrhœa ceases, the motions become semi-solid or even formed, and the mouth symptoms disappear, all except an unnatural smoothness and cleanness of the tongue. But the motions, though solid, are remarkably copious and do not regain their normal colour. They remain grayish white, pasty, acid in reaction and evil-smelling, and there is no gain in weight, perhaps progressive wasting. Sooner or later there is a relapse of all the previous symptoms, with increased emaciation, dry, scurfy skin and œdema, and the patient ultimately dies from exhaustion or some intercurrent disease.

Such is the progress of a badly managed or neglected case of sprue; but under judicious treatment the majority of cases, if taken early, recover completely. The motions become more and more formed and less copious, assume at first a pale, then a bright yellow colour, and gradually darken to a normal brown, and regain an alkaline reaction. This process occupies several months of careful dieting, especially difficult in a patient whose appetite is ravenous, whose imagination revels in thoughts of forbidden food, and whose will-power is weakened by long and exhausting illness.

Atypical forms.—The relative predominance of the intestinal symptoms, and the psilosis (of the tongue and mouth) varies, not only from day to day in the same individual, but in different cases throughout their course. In India the denudation of the tongue and buccal mucous membrane is, as a rule, less severe and constant than in Ceylon, the Straits Settlements and China, in which countries the state of the mouth has overshadowed the intestinal symptoms, as shown by the nomenclature adopted.

Sprue without diarrhœa.—The only feature present may be the passage of colourless motions. This may continue for long periods, without psilosis of the tongue, and without diarrhœa, the motions being formed, and often remarkably copious, and grayish white or faintly yellow in colour, with increasing debility and wasting, and generally a good deal of flatulent distension. The slightest indiscretion in food, or a chill will, in such cases, bring back all the characteristic symptoms, the liquid, frothy, white motions, the flatulence, and sore mouth.

Treatment.—For the successful treatment of a case of tropical diarrhœa, it is essential that the patient should be firmly impressed, first, with the gravity and possible fatality of his symptoms, and, secondly, with the promise which treatment gives of his recovery. Unless he accepts these two ideas it is extremely difficult to get him

to follow a tedious and monotonous treatment, which offers in all but advanced cases an excellent prospect of success, if carried out faithfully and patiently.

The two essentials of treatment are rest in bed and an exclusive milk diet. Even in apparently mild cases the patient should go to bed in a warm room, with an equable temperature, well aired, but free from draughts. He should wear a flannel binder round the loins and abdomen, and a warm knitted jacket covering the shoulders and arms. He should not be allowed to write letters or receive visits.

The milk should at first be given from 7 A.M. to 10 P.M. in doses of 6 ounces every $1\frac{1}{2}$ hours. In bad cases it should be taken slowly from a feeding bottle, or through a tube, or sipped with a teaspoon, and the process should occupy from 5 to 10 minutes, and it may be necessary to give two extra doses during the night. In this way the quantity taken in 24 hours is from 60 to 70 ounces, and after a few days it may be increased by 10 ounces daily, the amount of each dose being raised to 8 ounces, and greater quantities may subsequently be reached by shortening the interval to one hour, but not by increasing the amount given at each time. The rate of increase will be regulated by the power of the patient to deal with the quantity given, but ultimately, in favourable cases, $7\frac{1}{2}$ to 8 or 9 pints may be reached without discomfort, but it is not wise to force these amounts on a recent case, but to advance slowly in accordance with his increasing powers of assimilation.

In cold weather the milk may be warmed, but it generally is best borne and liked without any preparation. It may be boiled if the patient prefer it. As the gastric digestion is usually normal in these cases, no advantage is gained by peptonising the milk, except when the milk coagulates in the stomach in large masses, which afterwards give rise to pain and flatulence; peptonising prevents this, and sometimes does good in this way for a time. Koumiss may be substituted for a part of the milk, but it has no special advantage, and is not so sustaining as ordinary milk. The milk should not be diluted with aërated waters, which add to the bulk to be taken and increase the flatulence. If the patient likes the sharpness of the aëration, this may be accomplished without diluting the milk by means of "sparklets." In many cases the patients are unable to consume enough milk to sustain them, and cry out bitterly for more food. In that case the milk should be concentrated in an extemporised water-bath to half its bulk, and given in this concentrated form. In doing this the milk should be heated and not

boiled, but stirred the whole time to prevent the cream from rising and the formation of a skin on the surface (Thin).

Rest in bed should be enjoined as long as the motions remain loose, and the exclusive milk diet continued for at least six weeks after they have become solid. The patient should be made to understand these conditions when the treatment is commenced.

Improvement generally begins after a few days of this treatment, the first change being improvement in the consistency of the evacuations, a diminution of the flatulence, and the amelioration of the mouth symptoms, but any relaxation in the rigour of the treatment will be followed by a relapse. No addition to the milk should be allowed until the normal colouring matters have begun to appear in the stools. If the evacuations have become solidified and the patient be moving about, he will require more milk than when in bed.

With regard to drugs, opium and astringents are distinctly harmful; they may allay the frequency of the motions, and are therefore admissible in intercurrent attacks of ordinary diarrhœa (with coloured bilious stools), but they increase the flatulence and distress, and should not be given except in such circumstances. An occasional small dose of castor oil seems sometimes to be beneficial. In very recent cases, such as one sees in the tropics, half-drachm doses of liq. hydrarg. perchlor. three times a day seem to act like a chemical reagent. Under its use the motions become solid and of a bright yellow colour in forty-eight hours, and the patient goes on rapidly to recovery, but in more chronic cases, especially in England, the result is generally disappointing, but is worth trying when there is not a satisfactory response to the milk treatment alone. Yellow santonin has been used, and deserves a more extensive trial than it has received. To be of any avail it must have been exposed to sunlight till it has acquired a bright orange colour. The pale yellow santonin of the shops is usually not yellow enough and is ineffectual. It is prescribed in 5 gr. doses in a teaspoonful of olive oil once or twice a day for a week, milk diet and absolute rest in bed being at the same time enjoined. Yellow santonin has sometimes a very depressant effect on the circulation, and the first dose should not exceed $2\frac{1}{2}$ grains.

There are exceptional individuals who are unable to digest and assimilate milk in anything like the necessary quantity. In such cases it may be advisable to substitute or add the juice of 4 or 5 pounds of raw meat, with a little water to allay thirst, and by and by shredded, raw, or underdone meat may be given. In similar

circumstances the "Salisbury" treatment has been tried with success. This consists of from 2 to 3 pounds of lean meat free from fat, fibre, and gristle, taken in equal portions at intervals of six or eight hours, as mince or underdone chop or steak, along with 4 pints of hot water in twenty-four hours, given two hours before each meal and on going to and rising from bed. This diet is also useful when, after the milk treatment, every attempt to return to more ordinary food is followed by a relapse of the symptoms. In all severe and grave cases nutrient enemata should be employed to supplement the feeding by the mouth, if they can be retained. After the six weeks of solid motions on milk diet have elapsed some additions to the diet should be tried. It has been found that fruit, especially strawberries and stewed apples, may be given early, even while the diarrhœa continues, without ill effect, and it is very grateful to the patient. A little thin bread and butter and a raw egg added to the milk may be allowed, and boiled arrow-root, rusks, and malted foods should be tried; afterwards, and very cautiously, pounded boiled fish in the shape of "fish-cream," and, finally, some finely minced chicken; but during the whole of this time, milk to the amount of 3 or 4 pints daily must continue to be the principal form of nutriment. In many cases, six, eight, or more months elapse before the patient can be safely allowed ordinary diet.

Cases under treatment in the tropics should be sent to Europe as soon as the state of the motions makes this proceeding advisable, provision being made for the supply of fresh milk on the voyage. No case of this kind should on any account be allowed to go to "the hills" for change of climate. This change invariably confirms or aggravates symptoms of this nature. When a patient has completely recovered in Europe and has entirely regained his weight, strength, and ability to digest and assimilate a mixed diet, he may be allowed to return to the tropics, where he would probably not be specially liable to suffer from the disease. He should, however, be warned not to accept an appointment which would necessitate his serving in "the hills," because of the prevalence of "Hill Diarrhœa," which is so closely related to sprue, at high altitudes.

A. Crombie.

Hill Diarrhœa

A peculiar form of diarrhœa prevalent at elevations of over 6000 feet, characterised by copious, liquid, white, gray, or straw-coloured

motions, often frothy, and in typical cases confined to the early morning and forenoon, and sometimes occurring in epidemics affecting a large proportion of the adult population simultaneously.

Relationship to sprue.—A glance at the definitions of the two diseases will show that the clinical features of hill diarrhœa and sprue can be described in the same words. There is nothing to distinguish the appearance of the stools in hill diarrhœa from those of typical sprue, and they are produced at the same period of the twenty-four hours. The two conditions agree, moreover, in affecting only adults, and not infrequently the one condition passes into the other; but they differ in certain particulars which, in the absence of observations which would establish their identity in respect of their bacteriological cause, entitle hill diarrhœa to be regarded in the meantime as a distinct clinical entity.

The points in which they differ are the following: — Sprue originates in the plains, in islands, and at the sea-level; hill diarrhœa in mountains, at not less than 6000 feet of elevation. Sprue is always sporadic and of insidious onset; hill diarrhœa is seasonal and often epidemic, many individuals of a community becoming affected simultaneously, and suddenly, during periods of epidemicity. On the other hand, incipient cases of sprue on visiting a hill station where hill diarrhœa occurs invariably become at once aggravated, passing into a condition of confirmed, and perhaps severe sprue, which is not cured by a return to the plains. And certain cases of hill diarrhœa originating "in the hills" do not recover on returning to the plains, the symptoms continuing there and passing on insensibly into those of ordinary sprue. The two points last mentioned make for the identity of the two conditions; and the fact that individuals, who have resided in countries where sprue is endemic, may not manifest symptoms of the disease for several years after they have left the endemic area, suggests the possibility that hill diarrhœa does not originate in the "hills," but that those attacked by hill diarrhœa really harbour the germ of the disease before their arrival at high altitudes, and that it there finds conditions favourable for its multiplication. The immunity of children from both diseases is in favour of such a hypothesis, and the comparative immunity of natives from sprue, and their non-immunity from hill diarrhœa during periods of epidemicity, is not strongly opposed to it.

Pathology.—Nothing has been done in the pathology of hill diarrhœa, though the well-ascertained circumstances under which it occurs, and the fact that it is often epidemic, make it a much more

likely subject for successful research than its more insidious and sporadic congener; neither has it ever been positively ascertained whether the colourlessness of the motions is due, as in sprue, only to the absence of the bile-colouring matters, while the bile acids are abundant. The analogies of sprue would lead one to think, until this had been done, that the assumption that in hill diarrhœa the function of the liver is in abeyance is at least premature. The freedom from pain, and from blood and mucus in the motions, and the rapid recovery which so often immediately follows a descent to a lower level, prevent our acceptance of the theory that the condition is one of intestinal irritation or enteritis in any degree. Everything points, as in sprue, to its being due to the presence in the digestive tract of some organism which multiplies there, devours the colouring matter of the bile, and perhaps other constituents, and so allows of rapid fermentative changes in the intestinal contents.

The daily fluctuation of the symptoms—the restriction of the diarrhœa and other symptoms to the early morning and forenoon—is difficult to explain on any hypothesis.

Geographical distribution.—Hill diarrhœa occurs in all the hill stations of India, and especially in those situated in the Himalayas. It has been known in Simla, the summer quarters of the Government of India, ever since 1852, and is there familiarly known as "Simla trots." It is also exceedingly prevalent at Darjiling, as well as at Masuri, Naini Tal, and other stations whose elevation is 6000 feet or more. It is well known at Shillong and at Utacam and in the Nilgheries, and troops stationed in the higher parts of the Kyber Pass suffer in a similar way. It is also reported as occurring under similar conditions of elevation in South Africa, and it is even stated that the monks of the Hospice of St. Bernard (8115 feet) break down in health from a like complaint.

Seasonal and epidemic prevalence.—Although sporadic cases occur at all seasons of the year, and especially among new arrivals, hill diarrhœa does not become prevalent at any of the hill stations mentioned except during the rainy months of July and August, and a certain number of habitual sufferers from the affection are obliged to leave the "hills" every year during that period. It is also during, or rather at the commencement of the rains that epidemics occur. The Simla epidemic of 1880 anticipated the rains by five days, occurring at the end of a dry period of marked oppressiveness, of high temperature, falling barometer, and rising humidity. The first case was on the 12th of June, and during the next fortnight about 50 per cent of the adult population of Simla

were affected, three-fourths of the cases occurring during the first week. Natives suffered equally with persons of European extraction and habits. Children and adolescents under sixteen were exempt.

Symptoms.—The essential symptom is the decolorisation of the stools. This frequently occurs in new arrivals without any diarrhœa, but with an accompaniment of anorexia and flatulent distension; it may continue for many weeks, and it may happen each time an individual arrives from the plains. The same phenomenon is frequent after the subsidence of an attack of hill diarrhœa, and may continue for one or two months if the patient continue to reside in the hills; but the motions as a rule become at once normal in colour on return to the plains.

The more usual symptoms are, with or without a preliminary of ordinary diarrhœa, an imperious call to stool at or before daybreak, and the passage of a copious liquid motion, generally resembling white-wash in appearance and consistency, frothy, and accompanied with much flatulence. Between that time and 11 A.M. or noon there may be three or four or six, seldom more, exactly similar evacuations of the bowel. From the latter time till early next morning the patient is free from all symptoms, and is able to keep all his appointments and follow his usual avocations. Next day there is a repetition of the same copious diarrhœa in the early morning and forenoon, and subsequent freedom from trouble. The symptoms generally subside after a short time, but they may continue during the whole period of his stay in the hills, cease on his going back to the plains, and recur on his return to the high elevation next season.

The motions are sometimes of a pale buff colour, instead of having a milky appearance, and generally they are passed without pain or griping. Occasionally the rule of immunity after noon is transgressed, and there may be one or two motions in the afternoon or evening. The tongue is coated with a whitish or yellowish fur, and generally, though not invariably, there is much nausea and disinclination for food, especially in acute cases. The flatulence which accompanies the diarrhœa becomes more marked on the motions becoming formed, until they regain their natural colour, and until this happens there is a tendency to relapse of the diarrhœa.

If the symptoms continue there is necessarily much wasting and loss of strength, and if the case become chronic, denudation of the tongue (psilosis) occurs along with the other symptoms of confirmed sprue.

Terminations.—Many cases tend to recovery without special

treatment, apparently on the system becoming acclimatised to the conditions prevailing at high altitudes. Others resist all recognised methods of treatment, but recover on descending to the plains. If recovery do not take place as the result of acclimatisation, treatment, or return to lower elevations, the case is liable to pass into one of ordinary sprue, with psilosis, wasting, and all the other symptoms and sequelæ of that disease.

Treatment.—Except in cases of unusual severity it is not necessary to retain the patient in bed. He should be warmly clothed and his food should at first consist exclusively of milk, which it is often advisable to peptonise, a process which lessens the liability to the formation of curds and flatulence.

The ordinary treatment of diarrhœa by opium and astringents is of no avail in hill diarrhœa. Opium in any form may check the number and fluidity of the motions, but it increases the flatulent distension and discomfort, without in any way controlling the further progress of the complaint. Distinct benefit is obtained by the use of intestinal antiseptics, and experience shows that in this disease the best results are obtained from drachm doses of liquor hydrargyri perchloridi three times a day, about fifteen minutes after food. In many cases it seems to act within forty-eight hours of beginning its use. In all those cases in which diarrhœa is not a marked symptom, or which are not placed on peptonised milk, pepsin in 12 grain doses, or a corresponding dose of ingluvin, lactopeptin, and perhaps pancreatin, act most satisfactorily, and should be continued as long as the motions remain deficient in colour, though firm in consistency, along with the liquor hydrargyri perchloridi in smaller and less continuous doses.

If in spite of this or similar treatment the symptoms continue, and the patient's health begin to suffer, he should be urged to return to the plains, especially during the rainy months.

A. Crombie.

PERITYPHLITIS

By this term is meant a localised peritonitis in the neighbourhood of the cæcum and appendix secondary to some lesion of these structures, much more commonly of the latter, from which circumstance the disease is usually, though not altogether accurately, known as APPENDICITIS, which is here taken to signify inflammation of the mucosa of the appendix only, comparable to gastritis, enteritis, or colitis.

Etiology.—The infinitely greater liability of the appendix to originate the malady is chiefly to be explained by certain structural peculiarities of this organ, which favour the occurrence of inflammation and subsequent extension to the peritoneal coat. Of these may be mentioned the complete investment by peritoneum and the existence of a meso-appendix which is very variable in the length of its attachment to the gut, in many instances not reaching the tip, thus causing the tube to be twisted or bent in itself, and favouring its strangulation and the compression of its vessels. The lumen of the appendix communicates with the cæcum by an excessively small opening, adequate for the escape of the normal mucous secretion, but as a rule incapable of admitting such foreign bodies as are frequently alleged to gain entrance, and the difficulty is further increased by the aperture being "hidden away under the loose folds of mucous membrane which surround it"[1] (p. 8).

Reference has been previously made (p. 13) to the gaps in the muscular tissue of the appendix, allowing of continuity between the submucosa and the subperitoneal connective tissue, and also to the large amount of lymphoid tissue contained in the walls of the tube (p. 287), the function of which may probably be the production of phagocytes. As with the lymphoid tissue elsewhere in the intestine, it is best developed and more active in early life, disappearing somewhat as age advances. Definite lymph channels are also to be found in connection with this tissue, which still further facilitate communication between the mucous and peritoneal surfaces. Lastly, as a "remnant," useless and without function, it is unduly liable, like similar structures elsewhere, to become the seat of inflammation or of new growths.

[1] *Hunterian Lectures*, R.C.S. Eng. (1903), on the "Anatomy and Pathology of the Vermiform Appendix," by W. M'Adam Eccles, M.S., F.R.C.S.

Although for these reasons perityphlitis in the great majority of cases is secondary to disease of the appendix, nevertheless cases of cæcal origin do occur, and a similar localised peritonitis has been found in connection with other parts of the canal, *e.g.* the hepatic flexure of the colon (Treves). As a rule, however, lesions of the gastro-intestinal tract, elsewhere than the cæcal region, which permit of infection of the peritoneum, lead to a general peritonitis. Even some attacks which appear to have arisen from the cæcum should be referred to the appendix, when this is coincidently affected.

The immediate exciting cause of the peritoneal inflammation is the invasion of the serous cavity by micro-organisms. Of these the most constantly occurring (94 per cent) is the B. coli communis, but others, such as B. pyocyaneus and prodigiosus, streptococcus pyogenes, staphylococcus pyogenes aureus and albus, and the diplococcus pneumoniæ are seen. B. tuberculosis and B. typhosus have also been met with as well, and some of these have very rarely been found without the B. coli, which is a normal inhabitant of the intestines. Under certain altered circumstances, among which are unhealthy conditions of the mucosa and contents of the appendix, and possibly the presence of pyogenic organisms, the B. coli would seem to assume virulent characters, and so determine an inflammatory process, in the course of which excessively potent toxins are produced (p. 64).

The conditions which predispose to these results, by morbidly affecting the bowels, and facilitating the passage of the organisms from the gut into the peritoneum, which have been met with in association with perityphlitis, are :—

I. Inflammation of mucosa of appendix or of cæcum, or of both—

i. Catarrh. ii. Suppuration.
iii. Ulceration and erosion.

a. Catarrhal ; *b.* Stercoral ; *c.* Traumatic.
d. Tuberculous ; *e.* Typhoid ; *f.* Dysenteric (?).

II. Lesions special to appendix and frequently resulting from inflammation—

i. Gangrene ; consequent on intense inflammation, or strangulation of process.
ii. Stricture and occlusion, with cystic dilatation and retention of abnormal secretions, fæcal matter, or foreign bodies.

III. Actinomycosis of appendix.

IV. Malignant growths of cæcum or appendix.

V. Injury; blows or strains, especially if there be any disorder of the appendix.

These several morbid states have been elsewhere described (*vide* pp. 290 and 310), and here it will suffice to repeat that actual perforation of the gut is not requisite to set up infection of the peritoneum, since this has frequently occurred when the appendix presented to the naked eye a normal appearance, though microscopic examination has disclosed erosions of the surface and organisms throughout the thickness of the wall.

One of the most frequently occurring results of appendicitis, or it may be of kinking of the process, as it is also one of the most serious, is the retention within the appendix, which may be in consequence dilated, of morbid secretory and other products, affording as they do an excellent nidus for the development of pathogenetic organisms. Among these products are to be reckoned the concretions so often met with. These, consisting chiefly of phosphates and carbonates of lime and magnesia, are formed *in situ* from the secretions of the mucosa, occasionally with a particle of extraneous matter as a nucleus, and sometimes with a slight fæcal admixture, and are comparable to the enteroliths and rhinoliths found elsewhere. They may attain a size of an inch in length, but are usually much smaller, smooth, or with a roughened surface; they may play an important part in causing ulceration by the irritation to which they give rise, intensified as this may be by the presence of virulent germs. Closely resembling, as they often do, seeds, pips, or fruit stones, an erroneous impression has prevailed as to such bodies being the essential cause of the disease. It is very doubtful if such is ever the case. If, instead of partial closure with cyst formation and consequent retention or empyema, the lumen should be completely obliterated throughout by cicatricial tissue as a result of the inflammatory process, a natural cure of the appendicitis may be established, although the patient may subsequently suffer pain and discomfort from traction on surrounding adhesions.

As a determining cause of the unhealthy changes within the appendix the accumulation in the cæcum of large quantities of ill-digested and indigestible food stands almost first, and a large proportion of attacks follow on a full meal of such material, especially if taken when in a state of fatigue after a long abstinence,

or if there be any coexisting constipation, or chronic dyspepsia. Experiments on animals have demonstrated the great importance of retention of the contents of the appendix as a cause of perityphlitis.

Age.—Perityphlitis is to be met with at all ages, in infants and in the seventh decade; the greater number, however, are between the ages of ten and twenty-five, decreasing in later life, in part doubtless from the natural tendency to closure of the lumen.

Sex.—The majority of patients are males, some say 80 per cent. A partial reason for this may be in the fact that in females an additional blood-supply to the appendix is derived from the ovarian artery, whereby the risk of gangrene is diminished. On the other hand, owing to communication existing between the lymphatics of the right ovary and Fallopian tube with those of the appendix, infective disorders of the former may extend to the latter organ, though oftener probably in the reverse direction. Moreover, pregnancy in a woman who has previously suffered from appendicitis, and recovered without operation, causes a greater risk of perityphlitis in consequence, and this usually leads to miscarriage and death. Attacks also are not infrequently connected with menstruation.

Heredity.—The disease has been frequently observed in several members of a family, especially in those of the same generation.

Season and climate.—Statistics show that it is more prevalent in the summer and in hot climates.

The possible *relation* of the malady *to gout and rheumatism* has been discussed on pp. 286, 287.

Morbid anatomy.—"*The peritonitis* which is the basis of the affection may be of any degree and of any extent. It may be general or local, suppurative or non-suppurative; or a localised suppurative peritonitis may be associated with a diffused non-suppurative form of peritoneal inflammation" (Treves). Except in the very acute cases, which are quickly fatal, the peritonitis is limited by adhesions which mat together coils of intestine and omentum, the thickening of which from congestion and œdema adds to the palpable swelling. The differences met with on opening the abdomen in this disease depend, no doubt, on the nature of the infection and on the resisting power of the tissues of the patient. Thus a widespread acute peritonitis originating from the cæcal region is seen in the fulminant cases which last but a few hours from the commencement of the attack. But however mild the case may appear to be at first, the possibility of a general peritonitis, which makes the malady so serious, is always to be remembered. Or a localised abscess containing several ounces up to several pints of

pus may be seen, usually ill-smelling—the cavity sometimes containing gas which has resulted from decomposition. Or the inflammatory fluid may be very small in quantity, serous or sero-purulent, and of a greenish colour and fœtid odour; or there may be actual fæcal matter. In those cases in which frequent recurrences have happened and there has been no obvious suppuration, dense, firm adhesions are formed, binding the structure firmly down in the iliac fossa, in the interstices of which a bead of pus may be discovered. Equally variable is the condition of the appendix itself. As already said, it may give little naked-eye evidence of being diseased, though microscopic examination reveals catarrh of the mucosa, erosions and infiltration with organisms through to the serous surface. Or there may be distinct perforation of the appendix or of the cæcum, and a concretion may be found in the abscess cavity; or the appendix may be partly or entirely gangrenous and represented by a loose slough; or the distal extremity may be swollen and contain an unhealthy fluid, the tube being kinked or twisted on itself; or again it may be inseparably embedded in the fibrous adhesions.

As regards the *suppuration*, this may come on within forty-eight hours of the commencement of the attack, or may not appear for a fortnight or more, during which time the inflammatory process continues with fluctuating intensity. The resulting perityphlitic abscess may be loculated, or may burrow in various directions, into the pelvis, or the loin, or even across the abdomen to the opposite side; it has been known to extend upwards in the sheath of the psoas muscle to the spine, or behind the ascending colon up to and through the diaphragm. If not interfered with surgically and the patient survive, the abscess may burst through the abdominal wall above Poupart's ligament, or into the cæcum, or the general peritoneal cavity; much less often into the rectum, bladder, or thorax; and in very rare cases has been found to have entered the hip joint, or the pus passing down into the thigh has simulated a psoas abscess. The comparative frequency with which an intraperitoneal abscess originating from disease of the appendix may make its way into the cæcum has given rise to an exaggerated and erroneous idea of the frequency with which the cæcum is primarily involved in these cases. Suppurative pylephlebitis may be met with in association with perityphlitis.

Symptoms and course.—It must now be admitted that whatever may be the condition of the appendix—or in rare cases the cæcum—it cannot be said that perityphlitis (appendicitis so-called) exists until the pericæcal peritoneum is inflamed. Very considerable

disease may be found in the appendix—appendicitis, catarrhal and even ulcerative—more even than may be met with in a severe attack of perityphlitis, without the patient complaining of any symptoms referable to the cæcal region. But there are also a large number of persons who without suffering what is properly an attack of pericæcal peritonitis, yet have constantly experienced pain and discomfort in that region (sometimes, though erroneously, termed appendicular colic), sufficient may be to induce vomiting and causing very definite illness for a few hours, then completely subsiding, though recurring in some cases with sufficient frequency to seriously affect the general health. In other patients the subsidence of the attack is only partial, that is to say, a more or less persistent pain, discomfort, and tenderness are complained of, frequently extending to the back or down the thigh, with more severe outbursts at irregular intervals. Such individuals are never well and complain of various disorders of digestion, constipation, colicky pains, and an altogether impaired health. The numerous cases comprised under these groups, differing as they do in severity of symptoms, are nevertheless scarcely the subjects of perityphlitis, but rather of true appendicitis, since there are found various morbid changes in that organ of an inflammatory character, but without involvement of the peritoneum. They are to be contrasted with those cases where quite as much disease is to be seen, but without any coincident symptoms. What determines this symptomatic difference cannot be as yet explained.

Some observers have described a pain and tenderness with palpable hardness in the region of the appendix, together with digestive disturbance, as a result of the involution of the mucous and lymphoid layers of the organ and their conversion into contracting connective tissue, which to some extent normally sets in after middle life and occasionally earlier.

The symptoms properly referable to actual perityphlitis exhibit, as might be supposed, a great variety in severity as also in mode of onset, duration, and termination, so much so that no single account can be given that shall be descriptive of all cases. The following well-marked groups may be recognised, which include most cases met with, although some run an unusual course and cannot be so classified. It must, however, be understood that the category to which the case will be referred is not declared from the first, and is not to be determined by the character of the onset, for with but moderate symptoms at the start the gravest condition may ensue, and, on the contrary, a commencement marked by suddenness and considerable severity may prove to be but a mild attack.

(*a*) In the most acute, severe, and rapidly fatal cases, not inaptly termed "fulminant" from the suddenness and intensity of the symptoms, a person in apparently perfect health becomes almost at once in a condition of the gravest danger, due to the onset of a perforative peritonitis which has been set up by the sudden escape through an ulcerated appendix of a concretion, or by a rapidly developed gangrene. The symptoms are those of perforation and of more or less general peritonitis, frequently with but slight indication of the seat of the mischief. A state of toxæmia is quickly induced, of which collapse and vomiting are usually marked features, with some abdominal pain, but not necessarily any rise of temperature, though it may reach 103° or higher. Death in such cases has been known to occur in thirty-six to forty-eight hours. They are for the most part first attacks, and the peritonitis is general rather than localised, though almost equally severe symptoms may set in in the course of cases that have lasted some days and are of but moderate severity.

(*b*) In the majority of cases the symptoms, at all events at first, are somewhat milder, though the pain may be considerable, and this with tenderness and swelling soon point to the cæcal region as the seat of the trouble. The nature of the case is therefore sooner apparent, although the course and outcome are uncertain. It is very seldom that the attack begins with a rigor, and pain is usually the earliest symptom complained of, waking the patient up, for the illness very commonly begins in the night, often after a heavy meal, and going to bed in the ordinary state of health. At the commencement the pain is frequently referred to the abdomen generally, or more about the umbilicus, but soon becomes localised to the cæcal region, which is tender, the tenderness being often at its maximum at M'Burney's point; at the same time it is necessary to insist on the fact that this is not an absolutely diagnostic sign, as is often supposed. Although the pain is variously described by different sufferers, it is always very severe, enough sometimes to induce collapse, and usually paroxysmal. It frequently radiates to the loin or down into the testis or labium and thigh, and sometimes is of considerable intensity in the left iliac fossa, sometimes involving the bladder and causing frequency of micturition. Nausea and vomiting usually occur quite early in the attack, but the latter is not severe and may be absent. As a rule the bowels are constipated, although diarrhœa has been observed in exceptional cases, and tenesmus very rarely.

Other manifestations of the malady are essentially those of peritonitis. Pyrexia appears from the onset and soon reaches

103° F. or higher, and runs an irregular course for eight or ten days, when it gradually or rapidly subsides; now and then it continues even for several weeks. "There is very little relation to be observed between the temperature and the local manifestations in the iliac fossa; the swelling may diminish when the fever is high, or it may increase with no rise in the temperature" (Treves). The tumour in the cæcal region is often not to be felt for some days on account of the tenderness, which may be extreme and entirely prevent a satisfactory palpation. The general swelling of the abdomen, and especially the rigidity of the abdominal muscles, both of which start from the right iliac region and spread more or less over the belly, and are very marked manifestations, also interfere with the detection of the tumour at first. When recognised it is of variable size, generally elongated and more or less parallel to Poupart's ligament, the inner border being often very distinct, with a dull note elicited by percussion over it; or it may appear but as a mere sense of increased resistance in this region. It is due to an inflammatory thickening of the parts with some effusion, and usually also to a certain amount of fæcal accumulation in the cæcum. It is subject to great variation in size and distinctness, which is quite irrespective of the actual seriousness of the case. Digital examination per rectum may show an extension of the swelling into the pelvis.

The general condition of the patient is that of septic fever, anorexia, and thirst, with some headache and a sense and aspect of illness, the features being drawn, and the aspect being that known as "abdominal," very much in proportion to the extent of the peritonitis. The decubitus is dorsal and the knees are drawn up. The pulse, respiration, and urine are such as are usual in fever, but not specially characteristic. A certain degree of anæmia is usually to be observed, due to absorption of the infective products, and if not perceptible in the appearance of the patient is to be found on examining the blood, when the percentage of erythrocytes and of hæmoglobin are both diminished, the latter especially so. As the toxæmia becomes more intense the prostration deepens. Œdema of the right leg may result from pressure of the tumour, or thrombosis may occur in either leg. Pylephlebitis, hepatic abscess, parotitis, and epididymitis have been met with as very rare complications. In cases of this category where resolution takes place, improvement begins after six or eight days, and all acute symptoms have subsided by a fortnight, and convalescence is established by the end of a month, although some resistance and swelling may be felt

on deep pressure over the cæcal region for some time longer. A considerable proportion, however, of such cases proceed to suppuration and the formation of an intraperitoneal abscess. Whether a case will take this course it is impossible to say at the outset, and indeed may be difficult to affirm even when pus has formed. There are certain indications, however, that such a condition is in progress which should be carefully looked for, as its early recognition in view of operation is of the greatest importance. As already stated, suppuration may set in as soon as forty-eight hours from the commencement of the attack, or not for two or even three weeks; most commonly, however, it appears after the fifth or sixth day, should the case take that course.

The points suggesting suppuration are :—

(i.) A continuance of the symptoms after the sixth day, although it occasionally happens that the signs and symptoms may persist to a marked degree for much longer, and finally resolve, leaving nothing beyond some thickening of the appendix. A partial subsidence of the symptoms, followed by a sudden exacerbation between the seventh and tenth days, is very suspicious of the presence of pus.

(ii.) An increase in severity of the symptoms generally or of some special features. Thus a very large tumour, especially if early and quickly developed, suggests the probable formation of abscess, though not invariably so; and gas in the pus cavity may prevent dulness on percussion. The extension of the pain into the genitals or into the thigh, which is drawn up, occurs more often in suppurating cases, and the same may be said in respect to vomiting and diarrhœa.

The higher temperature points in the same direction, whether this be continuously higher, or higher after a fall about the sixth or seventh day. A rigor seldom marks the onset of suppuration, and is of no diagnostic value.

Provided temporary causes be excluded, a marked increase in the rapidity of the pulse, especially a rate of 120 and over, is of very considerable significance, and the more so if this increase be out of proportion to the temperature. The patient becoming progressively worse in general condition, a deepening prostration, and a dry and brown tongue, all help to suggest suppuration.

(iii.) Some help to the diagnosis of pus formation may be obtained by a blood-examination for the presence and degree of leucocytosis. A very high grade of this condition may be developed, the leucocytes amounting to 30,000 and more; but though this

occurs in suppuration, the same has been met with in gangrene of the appendix without suppuration, and even to some extent in mild cases which have soon cleared up. "Virulence of the toxin and the resisting power of the individual have much to do with the degree of leucocytosis. Generally speaking, the more intense the poison and the greater the resistance—as in young and previously robust individuals—the higher will be the leucocyte count" (M'Adam Eccles, *loc. cit.*).

But it cannot be said that any single circumstance will denote suppuration with certainty, and just as an extensive abscess may ultimately be found, the formation of which was unsuspected, so the most suggestive indications may be fallacious. Fluctuation in the tumour and redness and œdema of the integuments over it may be taken as conclusive as to its abscess character.

(*c*) A third group of cases included within the term perityphlitis is of the nature already referred to on p. 290 as being probably really an inflammation of the cæcum without involvement of the peritoneum. Occasionally, however, the case does proceed to suppuration, the starting-point of which is probably the appendix, although perforation of a cæcal ulcer may very rarely be the origin.

(*d*) It is estimated that the disease recurs in about 40 per cent of the cases in which the appendix is not removed after the first attack, or in which the structure has sloughed off, or the concretion escaped from the lumen of the process. And the attacks, similar to what has been described, may recur again and again, the probability of suppuration diminishing, though not altogether disappearing, with each, but the formation of adhesions continuously increasing. In women recurrences often occur with menstruation. When in the intervals apparent good health exists the condition is known as "recurrent" or "relapsing" appendicitis; when pain and tenderness persist with all degrees of ill-health it is termed "chronic." So long as the diseased appendix remains in the abdomen it is a constant source of danger, and any attack may prove fatal, or a progressive ill-health marked by much mental depression may be established, due to the constant pain and discomfort, together with serious digestive disturbances and a chronic toxæmia from septic absorption.

Diagnosis.—The recognition of this disease when fully established as a rule presents no great difficulty, notwithstanding the variability that obtains in respect to individual symptoms. But at the onset and for the first few hours, if the symptoms be very

severe, considerable doubt as to the real nature of the case may exist, since so many of the symptoms are common to many acute abdominal affections. Of these acute pain, often in the umbilical region, vomiting, collapse, and probably a high temperature, are the most constant. For this reason perforation of a gastric, duodenal, or other intestinal ulcer, even typhoid, may be mistaken for perityphlitis or the reverse, and if death ensue quickly a correct diagnosis may be impossible. Tuberculous peritonitis starting from the cæcal region, or peritoneal inflammation limited to the pelvis, may closely resemble perityphlitis. Intestinal obstruction, which may be a sequel of an attack owing to strangulation of the bowel by the adhesions which are formed, is taken for perityphlitis; the latter is frequently diagnosed in mistake for obstruction, especially ileo-colic intussusception in children. But the vomiting is more continuous and more profuse, the abdomen more rapidly and more fully distended, collapse supervenes more quickly, and the temperature is not so often raised in the obstructive lesion, and generally the evidences of obstruction rather than of peritonitis predominate.

The position and direction of the appendix are in some measure accountable for errors in diagnosis. When it is directed upwards towards the liver or right kidney, and gives rise to a perityphlitis, hepatic or renal colic may be diagnosed at the outset. It is usual, however, for the pain and tenderness soon to become sufficiently localised to show the real nature of the case, apart from the radiation of the pain towards the shoulder in gall-stone colic, and towards the testis with rectal and vesical tenesmus in the kidney affection. Hæmaturia and jaundice also are significant symptoms, as well as the severity and duration of vomiting, this being much the most marked in biliary colic, and may be absent in renal calculus.

Perityphlitis in children has been frequently confounded with disease of the hip joint with abscess. The position and limited range of mobility of the limb, and the pain being referred to the knee, are points to be investigated, as well, of course, as the swelling in the iliac fossa and the history of the case.

Perinephritic and psoas abscess may come to closely simulate a pericæcal suppuration, especially if there be any pain along the course of the ureter, and frequency of micturition, which are sometimes met with in the last-named affection. The spine and back should be carefully examined, and special notice taken of the absence of acute pain and gastro-intestinal symptoms, which tend to exclude the peritoneal lesion.

Among rarer conditions for which perityphlitis has been mistaken may be mentioned a movable right kidney, right-sided pneumonia, or diaphragmatic pleurisy, with acute pain referred to the abdomen (see Vol. IV. pp. 197, 259), cholecystitis and suppuration connected with the ovary.[1]

Reference may here be made to patients who become morbidly apprehensive of being the subjects of perityphlitis, the symptoms of which they have made themselves acquainted with, and so dwell upon their fancied ailment as to sometimes render it very difficult to be sure that there is not some ground for their complaint. Operation has been performed in such circumstances.

Prognosis.—This should always be regarded as doubtful and serious, since the mildest case may quickly become dangerous. It is estimated that at least 80 per cent of the fulminant cases are fatal, and that about 30 per cent of those in which an abscess is formed die; and since death seldom occurs in the mildest cases, it may be reckoned that 5 per cent represents the rate of mortality of all cases of all degrees of severity.

The probabilities of relapse in any case have been already referred to, and in estimating the chances of any individual case, the stage at which it is seen, the previous duration, the severity or otherwise of the local symptoms, and general condition and presence or absence of complications, have to be taken into consideration. Sir Frederick Treves lays "stress upon the fact that the great majority of cases of appendicitis recover spontaneously without either an operation or the formation of an abscess, that the ultra-acute cases are actually rare, and that relatively to the whole mass of examples of all degree suppuration may be said to be uncommon" (*Cavendish Lectures*, 1902). The usual causes of death are collapse or septic poisoning; or it may be the result of some complication such as pneumonia or pylephlebitis.

Treatment.—Something may be done in the way of prophylaxis, especially in preventing another attack in a patient who has once suffered and not undergone operation. And for this purpose strict attention should be paid to the avoidance of large meals and indigestible substances, to the proper mastication of the food, and to careful regulation of the bowels, for which purpose massage is peculiarly suitable. It may also be as well to keep the patient from time to time on a moderate course of some intestinal dis-

[1] For an account of some of these unusual conditions simulating appendicitis and peri-appendicular inflammation, see a paper by Dr. E. C. Janeway, *New York Medical Record*, 26th May 1900.

infectant, such as salol, or frequent small doses of calomel for a few days.

As regards the actual treatment of an attack, the question mainly turns upon whether operation should be resorted to, and if so, when. In the fulminant cases and those in which suppuration has taken place there can be no doubt. Prompt opening of the abdomen, and washing out the peritoneal cavity, and removal of the appendix, offer the only chance in the worst cases, and then owing to the profound toxæmia the result may be unsuccessful. Concerning the large number of cases which, though severe, are not of the very worst type, and where it is doubtful whether suppuration will take place, or even whether it have done so, much difference of opinion exists. Some, acting on the principle that the disease is due to an infective process, the course of which is hidden and not to be ascertained with certainty, advise an exploratory incision, with a view to further procedure should it be found necessary. "The alternative," it is claimed, is "to await events without being able to control them." But, as Sir Frederick Treves shows, "operations carried out during an acute attack are attended with a risk of life which is considerable, and is probably expressed by a mortality of over 20 per cent; while the removal of the appendix during a quiescent period is attended with a very trivial risk, which may be expressed by a mortality of 1 in 500" (*Cavendish Lecture*, 1902). Should the indications already discussed point towards pus formation, operation is obviously called for, and at once, but otherwise, so long as doubt exists, it is seldom advisable to take this step before the fifth day, and even then the condition of the patient and course of the symptoms must be carefully weighed, and the decision will after all in a measure rest with the personal judgment and bias of the physician and surgeon rather than on any hard and fast lines as to certain days and special symptoms.

When a case runs a course of ten days or more and ends without suppuration, the patient becoming convalescent, the question as to the propriety of operation will again arise, this time with a view to the prevention of subsequent attacks rather than the treatment of the present condition. Taking into account the large proportion of cases which recur, and the real danger which the existence of a damaged appendix—how far damaged being unknown—confers, it certainly seems advisable that a radical cure should be established by the removal of the affected structure, and this may be said to represent the general opinion at the present day.

Much the same considerations apply to relapsing appendicitis;

on the one hand is a condition which may at any moment prove serious, which is attended with more or less constant suffering and disablement and a progressive impairment of health, on the other an operation which is attended with less danger than is connected with an attack, and affording in most cases complete cure. It should be mentioned, however, that subsequently certain symptoms may develop, among which are a good deal of dragging pain in the cæcal region, often associated with movement of the bowels, due to the formation of adhesions which interfere with the intestinal peristalsis; intestinal obstruction from constriction of the gut by these adhesions; secondary abscesses caused by the retention of pyogenic bacteria, or of a fragment of the ligatures or dressings, or from the non-removal of the stump of an appendix after the abscess cavity has been drained; and fæcal fistula showing communication with the cæcum. The medical treatment of such cases as are clearly amenable to such procedure has been previously discussed (see p. 292). It is desirable to emphasise the fact that purgation should be moderate and not violent, and the opiate sufficient only to allay the pain and keep the patient quiet without being pushed to narcosis. The occasional benefit that follows the administration of salicylate of soda has been referred to (p. 287).

W. H. Allchin.

DISEASES OF THE PANCREAS

General Anatomy and Physiology

The pancreas, which is retro-peritoneal in position, lies for the most part transversely across the first lumbar vertebra, extending as far to the left as the spleen, which it touches, and lies upon the vena cava, aorta, thoracic duct, left pillar of the diaphragm, left kidney and suprarenal body. The right extremity (head), which is at an angle to the foregoing, lies in the concavity formed by the second, third, and fourth parts of the duodenum, with the common bile duct passing down behind. The superior mesenteric vessels emerge from the lower border, and the pancreatico-duodenal vessels cross over the front of the head. In front the organ is covered by peritoneum, and is separated from the posterior wall of the stomach only by the potential cavity of the lesser peritoneal sac ; the first part of the duodenum lies to the front and right of the head, "and the pylorus often touches it when the stomach is distended " (*Quain*); and the transverse colon with its mesocolon cross the lower end of the head.

The position of the pancreas in relation to the anterior surface of the trunk corresponds to the interval between two lines drawn horizontally across the left side of the middle line 3 and 5 inches above the umbilicus, being about the junction of the epigastric and umbilical regions.

When *in situ* the pancreas is irregularly prismatic on cross section, of about 8 inches in length, and is marked by the impressions of the contiguous organs ; on removal from the body it is flatter and somewhat longer. The average weight is about 3 ozs.

In general structure the pancreas resembles the salivary glands, though less compactly arranged. The lobules and lobes, which are separated by connective tissue, are composed of tubular and somewhat convoluted alveoli bounded externally by a basement membrane, lined by the columnar or polygonal secreting cells. These cells, which are nucleated and often contain a paranucleus, present an inner granular and an outer clear zone. During secretory activity the granules collect nearer to the lumen, and the entire cells become clearer than during rest, and longer. The ducts commence in the alveoli by extremely fine canaliculi which may be traced between, and even it is said into, the individual cells ; these are gathered up into the intercalary ducts lined by flattened epithelium, and then into larger channels lined by cortical and in the largest by columnar cells, finally forming the main duct or canal of Wirsung, which extends in the substance of the organ for its

whole length, ultimately joining with the common bile duct to form the ampulla of Vater which opens into the duodenum. Besides the proper secreting substance of the gland, there are to be found in the interlobular connective tissue isolated groups of cells—the cell islets of Langerhans—resembling those of the carotid and coccygeal glands, arranged in columns, and possessing an abundant blood-supply. They appear to have no relation to any ducts.

The blood-supply of the organ is set forth on the table, p. 523; the lymphatics pass to some of the cœliac glands, and the nerves are derived from the solar plexus.

The *functions* of the pancreas are—(i.) to form a secretion to a daily amount that is estimated at about 7 ozs., which is poured into the intestine, where it plays an important part in digestion, as previously described (p. 23); (ii.) the exercise of some action on carbohydrate metabolism by the means, it is presumed, of an internal secretion (Vol. II. pp. 148 and 180). Whilst it is certain that diabetes is a result of the experimental removal of the entire pancreas in animals and of a number of destructive diseases of the organ in man, though this is not invariable, it is by no means clear how this metabolic perversion is determined. Whether the hypothetical internal secretion antagonises some equally hypothetical substance that normally tends to cause an excessive conversion of glycogen into sugar, or whether this secretion aids in the complete decomposition of the sugar which is normally found, and that therefore accumulates in the blood, and so in the urine when the secretion is absent, is at present unknown. It is further supposed that the sources of the internal secretion are the cell islets of Langerhans.

General Etiology of Pancreatic Disease

It must be admitted that diseases of the pancreas, at least so far as they are clinically recognisable with any certainty, are of comparatively infrequent occurrence. It is, however, probable that the functions of the viscus are often disturbed, in common with those of other organs, as the result of some widespread morbid state, but in the resulting complex of symptoms those specially referable to the pancreas are, with our present knowledge, indistinguishable.

The chief causes of disease of the pancreas are:—

Trauma.—Contusions, lacerations, and rupture of the organ as the result of mechanical injury are occasionally met with. Being run over by a cart-wheel, blows or kicks in the abdomen, and bullet wounds, are the usual accidents. The situation of the organ affords considerable protection from injury, and when this has been severe enough to damage the viscus, neighbouring structures

are most likely to be involved, and contribute to the usually fatal result which is preceded by shock and collapse. The rupture of the organ commonly leads to extensive hæmorrhage. In those cases which recover the relation of the injury to the subsequent development of so-called "pancreatic cysts" is of interest.

Infection.—Inflammatory affections of the organ are known, both clinically and experimentally, to be the sequence of certain infections, of which typhoid and influenza are probably the most frequent. Similar changes may also develop in the course of septicæmia and pyæmia, and the pneumococcus has been reported as the cause in a case of suppurative inflammation. The most constant microbic associate of acute pancreatitis, however, is the B. coli communis, pure cultures of which have been obtained from the inflamed organs, the channel of infection being doubtful, though probably *viâ* the lymphatic spaces, and sometimes by the main duct.

The organisms of tubercle and more rarely of syphilis, the latter chiefly as a congenital manifestation, have also been known to produce their characteristic lesions in the pancreas.

It is doubtful how far *alcohol* is directly responsible for pancreatic disease, although its predisposing influence is most probable, whether by affecting the general nutrition of the tissues, and so rendering them more vulnerable, or by determining gastro-duodenal catarrh, from which the ducts of the gland may be involved by extension. In common with other organs the pancreas undergoes marked degenerative changes in phosphorus poisoning. Overdoses of the active principle of the suprarenal body (adrenalin) also cause destruction of the cell islets of Langerhans and produce glycosuria.

Secondary to disease elsewhere.—The vascular relations of the pancreas *viâ* the portal circulation to the liver and thence to the heart render the organ frequently liable to become chronically congested, whether from hepatic disease, obstructing the portal circulation, or from cardio-pulmonary disease of an obstructive character.

In like manner the continuity of the lining membrane of the pancreatic duct with the mucous membrane of the duodenum, and the common opening of the duct with that from the liver and gall-bladder, offer an opportunity for extension of an existent catarrh from the bowel to the pancreas, or for the passage of septic organisms from the duodenum along the duct. Occlusion of the duct, whether by calculus or by pressure from without, may lead to retention of the secretion with cystic and other changes. A re-

markable association between the presence of gall-stones in the common duct and pancreatitis has been pointed out by Mr. Mayo Robson, the explanation apparently being that under these circumstances bile finds its way into the pancreatic duct and sets up a hæmorrhagic inflammation, such as may be induced by the artificial injection of bile into the duct.

By its proximity to other structures, especially the stomach, post-peritoneal lymphatic glands, left kidney, liver, and colon, diseases of these organs may involve the pancreas by extension.

Age and sex appear to exert no special influence in the causation of pancreatic disease; although acute pancreatitis is far more common in adult males, and the prevalence of calculi in the same sex is still more marked, contrary to what obtains in respect to gall-stones. New growths have been met with at all ages from infancy to later middle life.

Lastly, it is to be observed that well-defined cases of pancreatic disease occur for which no cause can be assigned.

The Morbid Anatomy of the Pancreas

The structural changes, gross and microscopic, met with in diseases of the pancreas are numerous, and for the most part well defined, but in their detection some allowance has to be made for post-mortem changes due partly to natural decomposition processes, and partly to the digestive action on the tissues of the pancreatic secretion. It is the appearance of the glandular epithelium that is most liable to alteration by these conditions. As illustrating the frequency with which morbid changes of the organ are met with post-mortem, Dr. Hale White (*Guy's Hospital Reports*, vol. liv. p. 189) found that in 6708 autopsies made in the fourteen years 1884 to 1897 the pancreas appeared to be diseased or injured in 142 instances, that is, in 2 per cent of all cases dying in a large general hospital.

Vascular changes.—The liability of the organ to *chronic congestion* from portal obstruction has been referred to, and the effect of such disturbed vascularity is seen in hæmorrhage into the gland substance, which is, however, far from common, and in the production of fibrosis. The bleeding usually occurs in small areas, in some of which it is seen to be recent, as in others the presence of altered blood, pigment, and crystals of hæmatoidin indicates that the effusion had taken place some time previously.

Hæmorrhage is also frequently associated with acute inflammation of the organ, rarely from altered blood states, and may also result from injury, and, it is said, from eclampsia. As a rule the effused blood is small in amount, but it may be very considerable, infiltrating the entire organ, and without any detectable rupture of a large vessel. The blood is not always confined to the glandular substance, but may escape at the surface of the organ into the post-peritoneal connective tissue, and thence extend between the layers of the mesenteries or around the left kidney, or be poured out into the cavity of the small omentum, the posterior layer of which covers the pancreas anteriorly. The hæmorrhage may be confined to certain regions of the gland, or scattered throughout the organ; and in proportion to the amount and to the time that has elapsed since it occurred will be the appearances presented by the hæmorrhagic area and the degree of enlargement of the viscus. It has been suggested that the arrangement of the vessels in the organ favours the occurrence of hæmorrhage, which there is little doubt is easily induced.

Emboli would seem to be very seldom lodged in the vessels of the pancreas, contrasting in this respect to their frequency in the spleen and kidneys. Vascular obstructions, whether embolic or thrombotic, would probably determine hæmorrhage into the organ.

Inflammation.—Inflammatory changes, *acute* or *subacute*, are well-recognised affections of the pancreas, probably always of infective origin. The true glandular tissues exhibit the usual appearances of inflammation, an increased vascularity, cloudy swelling, and granular degeneration with proliferation of the nuclei of the gland cells, and infiltration of leucocytes, together with micro-organisms, such as the B. coli communis. In a considerable number of cases of acute pancreatitis there is superadded to these conditions a variable degree of hæmorrhage—"beneath the capsule and into the interstitial tissue between the acini and around the ducts, as well as into the fat cells and intercellular spaces of the peri-glandular connective tissue" (Dr. J. H. Bryant, *Tr. Clin. Soc.* vol. xxxii.)—*acute hæmorrhagic pancreatitis*—and this may be so extreme as to involve the whole gland, which may become gangrenous, and has been found in this condition lying in a pseudo-cyst formed by the adhesion of adjacent parts, together with a quantity of dark fluid free from odour; or the organ may be reduced to a mass of slough that is discharged into the bowel. Considerable extravasations of blood may also be found in the post-peritoneal and peri-renal fat. In other cases the inflammation proceeds to

suppuration, which takes place either in numerous foci throughout the gland, or a single abscess, as large perhaps as an orange, results. Lancereaux (*Traité des maladies du foie et du pancreas*, 1898) records a case of pancreatic abscess due to pneumococcal infection. Suppuration of the pancreas from pyæmia is very rare. When the abscess is large the portal vein may be pressed upon and ascites produced. Rupture into the bowel or peritoneal cavity is an occasional termination. These morbid changes lead to a considerable increase in the size of the organ, which is very soft and altered in colour, being redder than normal, and when hæmorrhage has occurred presenting a dark, almost black-red appearance mottled with yellowish areas and streaks, the latter due to "fat necrosis," to be presently described. Grumous, blood-stained material is commonly to be found in the pancreatic ducts, but inflammation of the ducts is not at present to be differentiated from lesions of the gland substance.

The adjacent peritoneum in these cases is sometimes inflamed, but this condition remains local in distribution; should the lesser omental sac become involved large collections of inflammatory products mixed with necrosed tissue may be formed, which may burst into the stomach or bowel, and blood or bile-stained fluid is commonly found in the general peritoneal cavity both before and after death; no explanation of the escape of bile in this fashion has yet been forthcoming (see cases of acute hæmorrhagic pancreatitis recorded by Dr. J. H. Bryant, *Lancet*, 10th November 1900). The splenic and even the portal veins may become thrombosed, and secondary abscesses may form in the liver, especially in the suppurative variety of pancreatitis. In not a few cases biliary calculi blocking the common duct, or lodged in the ampulla of Vater, have been found in association with acute pancreatitis. This calculus obstruction appears to favour septic infection of the pancreas, *viâ* the duct, from the duodenum, especially if there be any catarrh in this section of the bowel, as is not infrequent when the bile is excluded. Their presence is important as affording an explanation of the recurrent attacks of colic and sickness which not infrequently precede the actual pancreatic affection.

In a case reported by Dr. Osler "the semilunar ganglia were swollen, the nerve cells indistinct, and there was an interstitial infiltration of round cells. The Pacinian corpuscles in the neighbourhood of the pancreas were enormously swollen and œdematous." In other cases the sympathetic ganglia have been found normal.

Chronic pancreatitis; pancreatic cirrhosis.—Next to malignant

growths in the gland, the greater number of morbid changes in the pancreas are comprised under this head. The essential feature of the disease is a condition of fibrosis, either uniformly distributed through the gland and penetrating between the cells, and involving the cell islets, or mainly interlobular. The former condition has been found to occur especially in association with granular kidney (Dr. Tylden, *Proc. R. Med. Ch. Soc.*, 26th January 1892). As a result of this change the organ is sometimes increased in size, though oftener smaller, harder, and of a paler colour than normal. Microscopically there is seen to be an increase in the connective tissue stroma, which overgrowth extends to the subperitoneal tissue in which the organ is enveloped, matting together the duodenum, pylorus, and hepatic flexure of the colon, which become adherent to the liver and gall-bladder. Consequent upon the pressure exercised by the new formation the glandular elements are liable to undergo atrophy or fatty degeneration, which may be even recognised with the naked eye by the yellowish spots to which it gives rise.

Here, also, the connection of a chronic inflammation of the gland with the presence of gall-stones in the common duct is undoubted. A frequent determining cause of fibrosis is chronic congestion from portal obstruction or obstructive heart disease. A similar induration of the gland, more or less localised, follows upon extension of inflammation from adjacent organs, such as gastric or duodenal ulceration, that has extended through to the pancreas which may come to form the floor of the ulcer.

A chronic suppurative inflammation has been met with, lasting, it may be, for months, and the sequence of an acute condition, or, as sometimes happens, kept up by septic infection from the stomach or duodenum by means of a communicating ulcer, or the result of pyogenic infection of cysts in the organ by means of the duct. Along with the pus formation in such cases is a fibrotic overgrowth.

Tubercle.—The pancreas may become the seat of tubercle in the course of a general tuberculosis, or be implicated in tuberculous peritonitis. Caseation and even breaking down into an abscess may follow, but the disease is generally very slightly developed and is distinctly rare.

Syphilis.—The pancreas seldom, if ever, exhibits any change as the result of acquired syphilis, but occasionally in the congenital malady a fibrosis of the organ has been met with, and rarely gummata.

Degenerations.—*Fatty degeneration* and *fatty infiltration* are of occasional occurrence. The two conditions usually coexist and

merge the one into the other. Venous congestion, senility, and general obesity appear to be the most frequent precursors. Alcoholism may be in some cases a cause, and the gland shows, along with the liver and other organs, acute fatty degeneration in phosphorus poisoning. In extreme cases the secreting cells are crowded with fat globules, and the connective tissue corpuscles of the stroma are similarly affected.

Fat necrosis.—The change which is designated by this term consists, in small areas of one to four or five lines in diameter, of necrosed fat of a dull, opaque white, yellow, or orange colour, composed of fat cells full of granular caseous material and sometimes rosettes of acicular crystals, which give the reaction of fat. The cell nuclei do not stain with the usual reagents. The adjacent adipose tissue is quite normal in character, and is occasionally marked off from the necrotic spots by a zone of minute engorged vessels and small cell proliferation. These areas are to be found in the substance of the pancreas itself, but more numerously in the neighbouring post-peritoneal fatty tissue and in the contiguous mesenteries and omentum, the appendices epiploicæ, and even as far removed as the pericardial and mediastinal fat. The exact cause of the change is still somewhat obscure, but it is frequently, though not invariably, found associated with various forms of pancreatic disease, especially pancreatitis, hæmorrhagic and suppurative, and malignant disease. It is said that the condition may arise independently of pancreatic disease, or at least of obvious structural lesion. The change essentially consists in a splitting up of the fat—a digestion—into glycerin and fatty acids, the former being absorbed and the latter combining in great part with lime salts. The condition has been brought about experimentally by ligation of the pancreatic duct, and the generally accepted view is that it depends upon the action of a fat-digesting pancreatic ferment that has escaped owing to the structural changes in the gland; the occurrence of the necrosis at a distance from the organ being explained by absorption and distribution of the ferment by the lymphatics, just as a pericarditis or pleurisy may extend from a peritonitis. By some it has been regarded as a sympathetic neurotrophic manifestation, though without any good ground for the hypothesis.

Lardaceous degeneration, chiefly of the arterioles, capillaries, and perivascular tissue has been described, but it is very rare, and still more exceptional is it for the gland cells to be implicated. The organ so affected is commonly cirrhotic or fatty, being paler, firmer, and heavier than normal.

Atrophy.—This is a by no means uncommon condition, whether as a part of a general marasmus, or as the local effect of pressure on the glandular tissue by excess of interacinous fat or of fibrotic contraction, or as the result of occlusion of the duct by calculi, the lobules gradually wasting and disappearing until the organ may weigh less than half the normal, or be reduced to a few fibrous strands. When the atrophy is associated with a fibrosis that pervades the gland, a condition results resembling atrophic granular kidney, which indeed may coexist. When the organ is involved in a general cachectic state, the gland is said to be of a cylindrical shape, and both cells and stroma share in the wasting; when the change is associated with diabetes, the cells and islets of Langerhans are atrophied, but an interstitial pancreatitis is also present, causing adhesion to adjacent structures; the gland is often of a brownish colour. Whether these differences are constant further observation is required to determine.

New growths — *Malignant.* — Although constituting the greater number of structural affections of the pancreas, they cannot be said to be very common. Dr. Hale White found them to occur in about 0.82 per cent of 6708 post-mortems from all causes. By others they have been reckoned to constitute about 6 per cent of all cancers.

Carcinoma.—By far the greater number of pancreatic neoplasms are of this type, the majority being primary, resembling in this respect the uterus and heart, and differing from the liver. It is estimated to be three times more frequent in males than in females, and, as carcinoma elsewhere, to be most prevalent between the ages of fifty and sixty.

It is usually of the scirrhus variety, the encephaloid form being very rare. The head only is much the more frequently affected, less often the body or tail, and very occasionally the entire gland.

Owing to the pressure exerted by the growth the pancreatic duct is often dilated and may become cystic, and a similar dilatation of the bile duct and gall-bladder with enlargement of the liver may result, though these changes are far from being constant. Another pressure effect of frequent occurrence is ascites from obstruction to the portal vein, and more than once the fluid effused has been noticed to be chylous in character. In marked contrast to the frequency with which gall-stones are found in carcinoma of the liver or bile passages, calculi very rarely complicate cancer of the pancreas. The association of fat necrosis has already been mentioned.

As in a fair proportion of cases the growth has developed by extension from neighbouring parts, such as duodenum or gall-bladder, so a pancreatic carcinoma may invade adjacent organs, usually the duodenum, into which it may ulcerate, and less often stomach, colon, or solar plexus. Secondary deposits from the primary growth are generally to be found in the lymphatic glands, especially those in the portal fissure, in the liver, in the peritoneum causing ascites, and in the lungs. When the organ is the seat of a malignant new growth it is usual to find dense fibrous adhesions firmly attaching it to all the structures that are contiguous.

Sarcoma.—Both primary and secondary sarcomatous growths have been seen in the pancreas, usually of the small round-cell variety, and sometimes melanotic. The malignant neoplasms which occur in the gland in earlier life—one has been recorded at the age of four years—are of this form. These growths may attain a very considerable size, 4 or 5 inches in diameter.

Lymphadenoma.—Growths in the pancreas of this class are very rare. Dr. Hale White mentions a case (*loc. cit.*) which died of Hodgkin's disease, and the centre of the organ contained a lymphoid tumour.

Cysts.—The conditions leading to the formation of cysts in the pancreas are varied, the most frequent being obstruction to the duct by calculi, by a gall-stone blocking the duodenal papilla, by pressure on the duct by new growths of the head of the pancreas, of the duodenum, or other neighbouring organs, or by occlusion of the lumen by chronic catarrh. In such circumstances the duct of Wirsung may become uniformly dilated, or irregularly swollen at intervals into numerous retention cysts of variable size. The contents of these cysts resemble more or less closely the pancreatic secretion in proportion to the duration of their formation, later becoming turbid, and even hæmorrhagic or muco-purulent. Very exceptional varieties of cysts in this situation are those which are congenital in origin; those in which the pancreas exhibits multiple cysts in its structure in common with other organs, especially the liver and kidneys, due, it would seem, either to vacuolation of cells or to expansion of lymph spaces, or to dilatation of the smaller ducts (vide *Path. Soc. Tr.*, 1885, vol. xxxvi. p. 20) and hydatid cysts.

There are also certain cysts, often regarded as of the pancreas, which are more correctly described as being outside that organ, the limiting structure being often the lesser peritoneal sac, or structure contiguous to the pancreas bound together by adhesions.

Such cysts may attain a very large size and hold many pints of fluid, which may be clear and possessing the digestive properties in a lesser degree of the pancreatic secretion, or, as more frequently happens, the fluid contains altered blood—see peritoneal sanguineous cysts, p. 512. No doubt some change in the pancreas is connected with the formation of these cysts in very many cases, whether this be injury which causes an escape of blood from the organ into the sac, or, as there is reason to suppose is sometimes the case, a cyst of the pancreas communicates with the lesser peritoneal cavity, and it may be that the extent of destruction of the organ is so considerable that ultimately only a small portion of the gland remains. It also seems probable that some of the cases of sanguineous cyst are really the result of a hæmorrhagic pancreatitis which has not proved fatal in the acute stage.

Calculi.—These are sometimes found in the ducts post-mortem, and have been known to be passed per anum during life, but they are not common, and occur chiefly in middle-aged males. Occasionally they are single, but oftener multiple, and there may be a hundred or more, and of a white, yellowish, or gray colour, soft, and ranging in size from mere sand to 2 inches in length, and of the bulk of a walnut. They may be smooth or rough, round, oval, or branched. They mainly consist of carbonate of lime and some phosphate of lime with organic matter, and now and then contain cholesterin; one case of oxalate of lime has been recorded.[1] It is to be observed that the first-named ingredient is not a normal constituent of the pancreatic juice, and its immediate source and the conditions determining its precipitation are not apparent. They are sometimes found in association with an obstruction of the duct and retention of the secretion, but this is not invariably so, nor are all cases of impervious duct complicated with calculi. Occurring as they frequently do with inflammatory states of the ducts, their origin may be due to infection by micro-organisms, as is probably the case with biliary calculi. Their presence is likely to be attended with atrophy or cirrhosis of the gland, which sometimes shows fatty change also, but it is remarkable that they are very rarely associated with cancer of the organ. Less often some form of acute pancreatitis may be set up. Biliary and renal calculi have been known to coexist with pancreatic concretions.

Anomalies of development.—An accessory pancreas is sometimes to be met with, in the wall of the upper part of the jejunum, of the duodenum, or of the stomach. A part of the head may be

[1] By Mr. Shattock, *Tr. Path. Soc.*, Lond. 1896, vol. xlvii. p. 101.

separated from the rest, forming the so-called "lesser pancreas." The tail may be bifid, or the duodenum may be completely surrounded by the gland tissue. A second duct—duct of Santorini—even larger than the main channel, may be found, and this often opens into the duodenum above the common orifice of the bile and pancreatic ducts. Occasionally a single duct communicates with the bowel distinct from the hepatic duct; and when the pancreas is displaced upwards the duct has been found opening into the stomach.

General Symptomatology of Pancreatic Disease

The general symptoms of pancreatic disease are scarcely so distinctive as the characters of the morbid structural changes might lead us to expect, or as the importance of the organ concerned might seem to warrant. No one of the various manifestations associated with disease of the organ is diagnostic of the pancreas being the seat of the mischief, and even combinations of symptoms frequently leave the diagnosis uncertain, and to be arrived at in great measure by a process of exclusion. The difficulty of accurate physical examination of the organ doubtless contributes to this result. In not a few cases the first evidence of existence of disease is found post-mortem. It is also true that evidence of disturbance of the digestive function of the pancreas is less obvious and less precise than are the manifestations of perversion of the internal secretory function of the gland. This is perhaps due to the fact that the action on the alimentary principles normally exerted by the pancreatic juice can be also carried out by the saliva, the gastric juice, the succus entericus, and the fat-splitting organisms in the intestines, whilst so far as is known the internal secretion is peculiar to the organ and cannot be formed by any other.

Subjective symptoms.—*Pain and tenderness.*—The actual occurrence of these symptoms, or their degree of severity if present, is most variable in pancreatic disease, even in the same form of malady in different patients. Thus many cases of malignant disease and of calculi may run their entire course without giving rise to any sensory disturbance, and an abscess of large size has been found after death occupying the head of the pancreas that had caused no pain and was quite unsuspected during life. In contrast to such cases, the inflammatory affections of the organ and the rare conditions of profuse hæmorrhage into its substance are likely to be attended with pain which is usually of the severest character, the

paroxysms being agonising in their intensity. Sometimes, however, it is much milder, and may be a dull aching sensation, though liable to exacerbations. Of whatever degree as regards severity, the pain is generally described as deep-seated, sometimes localised at the epigastrium, or more directly over the organ, sometimes more diffuse, and as a rule extending through to the back between the lower part of the scapulæ, or passing round the costal margin on the left side to the back or into the shoulder. It is subject to being influenced by movements or by the posture of the patient, and still more by the taking of food, and is often relieved by vomiting or by pressure on the upper part of the abdomen. A sense of faintness or of actual syncope or collapse usually attends the most severe paroxysms.

The existence of tenderness may be difficult to ascertain owing to the distension of the abdomen or the rigidity of the abdominal wall, but it is frequently detected, being most marked just above the umbilicus or midway between that point and the ensiform cartilage. It is evident that neither pain nor tenderness are in any way diagnostic of the existence of pancreatic disease, nor as serving to distinguish it from affections of neighbouring structures. In several cases of malignant growth in the organ implicating the semilunar ganglion and solar plexus severe pain and tenderness have been observed.

As in affections of most of the abdominal viscera, pancreatic diseases, particularly those of a chronic character, are likely to be associated with *mental depression*, low spirits, and despondency; but these are by no means constant, and it cannot be affirmed that when present they are directly due to the pancreatic lesion.

Dyspeptic symptoms.—Although there may be good reason to believe that the pancreatic secretion does not reach the bowel, and may be so proved post-mortem, it is difficult to assign any specially characteristic symptoms to that circumstance, unless it be the resulting emaciation and certain appearances presented by the stools and urine to be immediately referred to. And all important as the secretion is in the digestive process, its absence or alteration and composition cannot be said to give rise to any recognisable phenomena of a distinctly dyspeptic character; anorexia, nausea, vomiting, and diarrhœa, though frequently accompanying diseases of the pancreas, are by no means constant or peculiar to them. An excessive salivary flow, which was formerly regarded as symptomatic, is certainly not so, and it is more than doubtful whether any hypersecretion of the pancreatic juice occurs with regurgitation into the stomach and subsequent vomiting, as has been stated.

Characters of the excreta.—The diminution or complete absence of pancreatic secretion would be expected to affect the character of the fæces and also in some measure the composition of the urine.

(*a*) *Fatty stools.*—The occurrence of free fat in the motions in appreciable quantity has long been known as a frequent accompaniment of pancreatic disease. It appears as an oily film on the surface of the fæces, from which it separates soon after being voided, or as greasy, tallowy-looking globules and masses, or a crystalline material consisting of fatty acids and salts, and generally emitting a very foul odour. This condition has been noted in malignant disease of the pancreas, especially when the head is the seat of the new growth, and it has also been met with in other morbid states of the organ, though with no great constancy in any case. Its diagnostic value is, however, diminished by having been known to occur in diseases of the intestinal mucosa, when the absorbing power of the bowel is much impaired, and also in disease of the mesenteric glands.

(*b*) A considerable amount of undigested muscle fibre in the fæces has been held by some authorities to be indicative of pancreatic disease.

(*c*) Certain conditions of *colourless stools*, occurring without jaundice, have been ascribed to deficient pancreatic secretion in the intestine, this fluid being supposed by some to take part in the changes in the bile pigments which determine in great measure the colour of the fæces.

(*d*) *Lipuria.*—Except in association with filarial disease (Vol. II. p. 28) the presence of fat in the urine is very rare. Among the few conditions which determine it, pancreatic disease is to be included, others are fat embolism from fracture involving the bone marrow, phosphorus poisoning, and the lipæmia of diabetes mellitus.

(*e*) *Indicanuria.*—A diminution in the amount of indican in the urine is said to occur in pancreatic affections, owing to the lessened production of this substance in the intestines from suppression of the pancreatic secretion.

(*f*) Mr. Mayo Robson (*Brit. Med. Jl.*, 11th May 1901) has noticed with some constancy the deposition of "delicate yellow needles in sheaves and rosettes" in the urine of patients suffering from chronic jaundice and pancreatitis, when boiled with an oxidising agent and treated with phenyl hydrozone; no such crystals were found in cases of simple catarrhal jaundice or other morbid urines.

(*g*) Under normal circumstances salol, when taken by the

mouth, is decomposed in the intestine into compounds of phenol and salicylic acid, which are excreted in the urine as sulphocarbolates and salicylurates, and if in excess give a dark or greenish colour to the excretion. In the absence of pancreatic fluid from the intestine these changes do not take place. As a test, however, of pancreatic disease this cannot be said to be of much practical value.

Emaciation. — Even apart from diabetes there is a great tendency for pancreatic disease to cause rapid and extreme loss of flesh, probably by interference with fat and carbohydrate digestion, and this may be seen when there is no glycosuria and no malignant growth, although these conditions necessarily increase the liability. In estimating the significance of this condition it is to be remembered that disease of the pancreas very frequently supervenes in exceedingly fat people, and hence the loss of weight may not at first be quite so apparent. At the same time it must be admitted that cases of disease of this organ do occur in which the loss of flesh is only moderate or even slight, though the reverse is the more usual.

Diabetes.—Some account of the relations of pancreatic disease to diabetes has already been given in the description of the latter affection (see Vol. II. p. 180). Extended observation has shown that glycosuria may occur with any form of disease of the pancreas, though it is much the most commonly associated with atrophy or with chronic inflammatory changes and fibrosis of the gland, with or without calculi, and in these cases diabetes of a rapidly fatal course is usual. It appears to be necessary, however, for the establishment of this complication that the entire gland should be involved. Nevertheless diabetes is not of invariable occurrence even in such conditions, and it must of course be remembered that grave diabetes may develop when the pancreas is quite healthy in appearance. A special connection between bronzing of the skin and pancreatic disease, *diabète bronzé*, has been described, but it is certainly not common (see Vol. II. p. 189). From all this it is clear that the relationship of diabetes to disease of the pancreas is not a direct one, and various suggestions have been made to explain it, but so far not satisfactorily. Nor is this surprising, since the precise origin of the urinary sugar, whether from deficient oxidation of that normally formed, or from over-production, still remains undetermined.

A case reported by Dr. Byrom Bramwell (*Tr. Ed. Med. Chi. Soc.*, 5th Feb. 1902) would seem to suggest that a deficiency of pancreatic

secretion might be responsible for a more profound and general metabolic disturbance than is even represented by diabetes or emaciation, viz. the condition known as *infantilism*. A youth extremely backward in growth and development, who had suffered from chronic diarrhœa for nine years, and in whom by elaborate analysis the pancreatic secretion was shown to be deficient, was relieved of the diarrhœa and grew considerably under the administration of pancreatic extract.

Tendency to hæmorrhage.—Not only does there appear to be a remarkable liability for bleeding, sometimes of a profuse character, to take place into the substance of the gland itself apart from any mechanical congestion or other disease of the organ, and without the obvious rupture of vessels, but there also is, as Mr. Mayo Robson has pointed out (*Brit. Med. Jl.*, 11th May 1901), in cancer of the organ a great tendency to hæmorrhage from operation wounds and elsewhere, sufficiently severe even to cause death. It does not appear to be dependent upon the frequently associated jaundice, though probably increased thereby, and its pathology is very doubtful. The production of pancreatic hæmorrhage experimentally by the injection of various pathogenetic microbes suggests that it is due to some toxins; others, noting its frequent coexistence with fat necrosis, have connected it with the absorption into the blood of some ferment of the pancreatic juice. A marked diminution in the number of blood plates has been observed in such cases. A predisposition to thrombosis of the veins has been noticed in this affection, such as Trousseau affirmed to be generally the case in "cancerous patients."

Effects of pancreatic disease upon other organs.—The most important of these are due to the pressure of a pancreatic growth or cyst upon adjacent parts, or the formation of adhesions with neighbouring structures. Hence may be established jaundice by obstruction to the common bile duct, distinct from the existence of biliary calculi, which are frequent; ascites from pressure on the portal vein; œdema of the lower extremities from pressure on the inferior cava; hydronephrosis from compression of the ureter; disturbance of gastric action, and even dilatation of the stomach from adhesions between the organs or traction on the pylorus. By extension of new growths of the pancreas, the stomach, colon, duodenum, etc. may become involved, and vomiting, hæmatemesis, blood in the stools, and other symptoms may be produced.

The Physical Examination of the Pancreas

Inasmuch as the organ in health is seldom capable of being subjected to physical examination, for it is only in extremely thin people that it may be detected, the comparative data for its investigation when diseased are in a great measure wanting. General or localised enlargements may sometimes be recognised by palpation, and are characterised by being deeply seated with the resonance of stomach or colon in front. The state of distension of these organs, and still more the rigidity of the abdominal wall in the epigastric and upper umbilical regions, more particularly of the right rectus abdominis, offer considerable difficulty to the manipulation of the pancreas, and beyond being able to affirm that a given tumour is post-peritoneal, and in the situation of the pancreas, a more precise diagnosis is often unattainable. The relaxation of the parietes by the administration of an anæsthetic, and the emptying of the bowels by enema, may facilitate the examination. The extent to which the aortic pulsation may be communicated through a pancreas, whether normal or altered by disease, is very variable, and is of no special significance provided the distensile character of aneurysmal pulsation be excluded. The physical features of "pancreatic sanguineous cysts" are described on p. 513.

HÆMORRHAGE INTO THE PANCREAS

Symptomatology. — The manifestations of this condition, whatever its cause (p. 361), will chiefly depend upon the amount of blood that is effused. When this is slight its existence may be unsuspected, although post-mortem clear evidence of hæmorrhage may be apparent. When, however, the bleeding has been considerable, epigastric pain, usually sudden and most severe, is the most prominent symptom; sometimes it is more diffused and of a colicky character, and with it tenderness on pressure over the upper zone of the abdomen, with perhaps a sense of constriction round the lower part of the chest, may be complained of. Nausea and vomiting to a variable degree are present, and it is said also "an urgent desire for defæcation" (Leo, *XX. Century Practice of Medicine*, 1897). The general symptoms are those common to intra-abdominal injury from any cause, a sense of anxiety and dread, with shock and collapse, not necessarily proportionate to the hæmorrhage, but deepening to a fatal issue within a few hours, or it may be days. It is seldom, if ever, that the amount of blood lost is responsible for death, except in traumatic cases, when other organs

share in the damage and contribute to the bleeding. When, however, the hæmorrhage is extreme, the symptoms are rather those of sudden and increasing syncope, a pulse that is small, rapid, and becoming imperceptible, a lowered temperature, with pallid skin covered with cold sweat, and supervening unconsciousness, with an occasional convulsion. Both albumin and sugar have been found in the urine, which may be much diminished in quantity in severe cases of rapid onset and short duration. Such symptoms afford no sure ground for a **diagnosis** beyond probability, and, except that an internal hæmorrhage is occurring, nothing certain may be affirmed, the exact seat being doubtful. This, added to the almost invariably fatal ending, combine to prevent any rational **treatment**, which can only be directed to sustain the patient as long as possible. How far such a drug as adrenalin might be of use remains to be seen. Opening the abdomen and seeking to arrest the bleeding by surgical means has been proposed.

PANCREATITIS

Acute pancreatitis—**Symptomatology.**—Acute pancreatitis is essentially a disease of rapid onset, characterised by extremely severe symptoms of short duration and of grave import. It is probable, however, that milder cases do occur from time to time that recover, but the difficulties of precise diagnosis render their detection at the best uncertain; and in the severe cases a history of previous recurring attacks of colic and sickness extending over several years is sometimes to be obtained, possibly due to gall-stones. Although the inflammation may assume very different anatomical features—hæmorrhagic, suppurative, or gangrenous—these conditions are seldom to be differentiated during life, and the general symptoms are in the main the same whatever be the exact nature of the lesion. As a rule the patient, who has probably been in previous good health, or it may be with a long history of dyspeptic symptoms, is suddenly seized with severe epigastric pain, continuous in character, though marked by paroxysmal exacerbations. Movement increases the suffering, which may become so intense as to induce fainting and collapse, which a constant vomiting favours and intensifies. It is seldom that the pain is very localised, spreading as it does from the epigastrium over the upper zone of the abdomen. In rare cases, it is said, where the onset has been gradual, and suppuration ensues, pain has been

scarcely complained of. Extreme tenderness is elicited over the same region, especially on deep pressure, and a circumscribed tenderness over the pancreas with tender spots distributed over the abdomen, the seat of areas of fat necrosis, have been regarded by Fitz as of valuable diagnostic significance. The vomited material consisting of food and mucus, and often a green fluid from altered bile, or containing blood, is ejected after violent and distressing retching. Rigors are frequent both at the onset and later, when suppuration is taking place. Some degree of pyrexia is nearly always present, and this may reach to 104° F., with irregular fluctuations; and the pulse is rapid, small, and thready or soft and compressible. Another and almost invariable symptom is constipation. A troublesome hiccough may add to the distress of the patient, whose suffering is still further increased by a thirst which the vomiting prevents any alleviation of. The anxious expression and the pinched and drawn features, with a pallor of countenance and forehead covered with cold sweat, give rise to an appearance of severe illness, which is as real as it appears to be. After a few hours the restlessness, which is often to be noted at first, gives place to a state of prostration induced by the pain and vomiting and general sense of extreme illness. Not infrequently there is slight wandering and delirium. Some jaundice also may supervene, especially if an abscess form of sufficient size to press on the common bile duct. It is usual to find the upper part of the abdomen somewhat distended, and if the tenderness and the rigidity of the parietes permit of deep pressure being made, a sense of resistance may be experienced over the region of the pancreas, with the stomach resonance in front; a general tympanites may be set up.

The urine is scanty, and may contain albumin and casts, but very seldom sugar. If an abscess have formed, and have burst into the bowel, pus will appear in the stools, and gangrenous masses have also been known to pass per anum.

Although most cases of acute hæmorrhagic pancreatitis prove fatal in the course of two or three days from exhaustion, recovery has been known even when sloughing of the pancreas has taken place, and some of the milder cases have eventually died after a few weeks from septicæmia, especially those where suppuration has developed slowly. Others pass into a chronic condition, and terminate in death from diabetes.

Diagnosis.—The clinical picture of the disease differs very slightly, if at all, from that of an acute peritonitis of the upper part of the abdomen, with which indeed the pancreatitis is likely to be

complicated, and especially if suppuration have taken place. The diagnosis of acute pancreatitis has been made during life, but, as a rule, the actual nature of the malady is one of probability rather than of certainty. Attention has been drawn to the importance attached by Fitz, who has done much to elucidate the diseases of the pancreas, to the association of sudden epigastric pain, followed by vomiting, collapse, and constipation, with the subsequent appearance of a circumscribed swelling and tenderness over the organ, with tender areas elsewhere over the abdomen.

The symptoms often suggest intestinal obstruction, but their localisation to the upper region of the abdomen, where obstruction of the bowel is unlikely to occur, should serve to distinguish acute pancreatitis, which is commonly much more sudden in onset. Yet identical symptoms to those above described have been found in a case of obstruction of the ileum by a gall-stone, and also in acute membranous enteritis, limited to the end of the jejunum and first part of the ileum; but such cases are very unusual, rarer even than acute pancreatitis. The rapid commencement, together with the severe retching and vomiting, affords a close resemblance to irritant poisoning, although in this case diarrhœa rather than constipation is the more probable, apart from any help the history of the case may afford. In many respects the condition simulates an attack of biliary colic, particularly if there be jaundice; the situation of the pain in the right hypochondrium may afford a clue, although the progress of the case will soon assist in the diagnosis, whilst remembering the frequent association of gall-stones with acute pancreatitis. The abdomen has more than once been opened with the expectation of finding a perforated gastric or duodenal ulcer, which is probably the most usual cause of acute peritonitis in the upper abdominal region.

Treatment.—The disease is one for which but little can be done beyond endeavouring to allay the pain and vomiting by morphine administered hypodermically, with hot fomentations applied to the abdomen, and to avert the syncope and collapse by stimulants.

If there be reason to suspect that pus has formed, laparotomy is justifiable, but it cannot be regarded as a very hopeful procedure, having regard to the extremely serious nature of the disease.

CHRONIC PANCREATITIS — **Symptomatology.** — The morbid condition to which this term is applied, and that is represented anatomically by the condition of "cirrhosis," or "chronic interstitial inflammation," leading as a rule to an atrophy of the organ (see p. 363), is seldom a sequel of acute pancreatitis, but develops

slowly and insidiously with but few distinctive indications of the organ that is really at fault. The symptoms are essentially those of an increasing dyspepsia, and chiefly suggestive of gastric derangement, such as loss of appetite, nausea, flatulence, and distension of the upper part of the abdomen with acid eructations. Pain and tenderness are very variable in their presence and in their degree, a deep-seated discomfort being often complained of, that may amount to severe pain, which tends to pass through to the back or round the left rather than the right side, the tenderness being experienced over the region of the organ which may give rise, on palpation, to an increased sense of resistance in that situation. Mr. Mayo Robson, *loc. cit.*, who believes this "to be a much more common affection than the acute or subacute forms, and thinks that some cases put down as malignant disease of the head of the pancreas may be cases of chronic interstitial pancreatitis, has drawn particular attention to the occurrence of jaundice in this affection." Associated with the jaundice, which soon becomes persistent and of increasing intensity, are "paroxysmal attacks of pain and ague-like seizures," which give rise to a suspicion of gall-stones; but the situation of the pain is not that of biliary colic, nor is there tenderness over the gall-bladder, as commonly occurs with calculus, though it is distended. There are many cases, however, in which the structural changes of chronic pancreatitis are met with where obstructive jaundice is not present. Quite as important is the development of diabetes mellitus, which is a frequent though not invariable complication of a fibrotic pancreas. As with the other diseases of the organ that are associated with glycosuria, the extent to which the glandular substance is destroyed would appear to determine the presence or absence of sugar in the urine. Due to the dyspepsia, no less than to the diabetes, the subjects of chronic pancreatitis progressively waste as the general ill-health increases, which a diarrhœa may still further intensify. The stools, even in the absence of jaundice, are frequently pale or even colourless, owing, it is to be presumed, to some defective changes in the biliary pigments in their passage along the intestines. The close relation of the pancreas to the portal vein explains how a moderate ascites may still further complicate chronic pancreatic fibrosis, by leading to a contraction of the new-formed connective tissue with constriction of the vessel.

Diagnosis.—This is admittedly most difficult, and is seldom other than a probability. The presence of glycosuria and wasting, with pain and tenderness in the upper abdominal zone, would

strongly suggest chronic pancreatitis, which would be rendered more likely if any swelling or increased resistance were perceptible in the same region.

When icterus rather than diabetes is a marked feature, it is necessary to differentiate the condition from other causes of chronic jaundice, more especially gall-stones and malignant disease involving the main bile duct either primarily or by pressure thereon. "From gall-stones," says Mr. Mayo Robson (*Lancet*, 28th July 1900), "the diagnosis is of no great importance, since the two diseases are often associated, and the treatment is the same; nevertheless it may be possible to make a diagnosis of gall-stones by the sequence of a long antecedent history of spasms without jaundice, then by a severe attack of pain followed by jaundice, and after a time by recurring pains with increase of the icterus associated with ague-like attacks. The absence of tumour is more common in gall-stones than in chronic pancreatitis, though in the latter the gall-bladder may be found contracted at times. Although in pancreatitis there is usually less pain, the paroxysmal attacks may be equally as severe as in gall-stone seizures." Attention has already been drawn to the difference in the situation of the pain and tenderness in these conditions. As regards cancer of the head of the pancreas, it usually occurs later in life than the chronic inflammatory affections. "It is preceded by general failure of health, and when the jaundice supervenes it becomes absolute and unvarying, and is nearly always associated with a tumour of the gall-bladder which generally attains a large size and shows no tenderness on manipulation. In some cases of cancer of the head of the pancreas a hard nodular tumour may be found on the inner side of the distended gall-bladder. Extremely rapid loss of weight and strength, with increasing anæmia, but without ague-like seizures, is very characteristic, and it is common for there to be an absence of fever with a slow, feeble pulse, and later ascites with œdema of the lower limbs. . . . In many cases a diagnosis will only be made by an exploratory operation. . . . If ascites be present the tumour will probably be malignant. The lymphatic glands in the lesser omentum may be enlarged in both cancer and chronic pancreatitis, but in the latter they will be discrete and soft, in the former hard and perhaps infiltrating. Adhesions are usually associated with chronic pancreatitis, but are generally absent in cancer."

Treatment.—The first indication in the treatment of this disease is to so arrange the diet as to make the least demand

upon the deficient secretion of the organ. To this end fats should be reduced to a minimum. Provided there be no glycosuria, and even then it is inadvisable entirely to exclude them, no special limits need be placed on the amount of carbohydrate foodstuffs; whilst the proteid aliment must be maintained. It is often difficult to induce the patients to take sufficient food, and the appetite requires tempting. Partially digested foods, such as Benger's and the various malted foods and peptone preparations, minced raw pancreas, or the liquor pancreaticus (Benger), may be administered with distinct advantage, especially in the diabetic cases.

The most troublesome of the dyspeptic symptoms is the nausea, which may be relieved by a simple effervescing mixture containing hydrocyanic acid; and an acid and bitter mixture may be given.

Where the pancreatitis is associated with jaundice Mr. Mayo Robson has met with considerable and lasting success by drainage of the gall-bladder.

No special symptomatology characterises either TUBERCULOSIS or SYPHILIS of the pancreas, which are of pathological rather than of clinical importance. The latter, so far as it is recognisable during life, presents the symptoms of a chronic interstitial pancreatitis.

MALIGNANT DISEASE OF THE PANCREAS

Symptomatology. — The close anatomical relations of the pancreas to the duodenum, pylorus, gall-bladder, and other structures, as well as the liability of the gland to be invaded by new growths from these organs, render the symptoms of pancreatic cancer less distinctive than might be supposed. No little variation is also to be met with in the actual occurrence, or in the degree, if present, of symptoms which are associated with the development of malignant tumours in this situation, corresponding in this respect to what may be generally affirmed of diseases of the abdominal viscera.

In illustration of this may be mentioned *pain*, which, although present in most cases, is occasionally almost or entirely absent. Usually of a more or less constant, deep-seated, aching character, with paroxysmal exacerbations increasing in severity, it may be relieved by pressure, or, on the contrary, there may be extreme tenderness. Some measure of abdominal distress, of a feeling of sinking, of faintness, and anxiety, with the acquirement of the "abdominal facies," are generally experienced.

Owing to the depth of the organ from the surface, and still more to the condition of the abdominal walls, as well as the size of the growth or part of the gland involved, the recognition of a *tumour* is very uncertain. In favourable circumstances a deep-seated, fixed, firm mass may be detected—it is said to be felt in a quarter to half the cases—but more often nothing beyond a sense of increased resistance, midway between the umbilicus and ensiform cartilage, is to be observed. A noticeable indication of deep-seated mischief, such as that of the pancreas, is to be found, as originally pointed out by Jenner, in a marked rigidity of the upper part of the right rectus abdominis, sufficient to simulate a tumour and to interfere with palpation in that region. In front the percussion resonance of stomach or colon can be elicited. Lying as the swelling does over the aorta, a communicated impulse is sometimes very marked, and a bruit may even be audible over it. Very exceptionally, the tumour has been known to move with respiration.

The pressure effects of the growth, however, are more constant, and of these *jaundice* is the most important. When the head of the pancreas is the seat of the cancer, as it commonly is, icterus is all but invariable and of a deepening intensity, and absolutely persistent after its first appearance. The colour of the skin passes from a bright to a more orange yellow, and finally to a dark green tint. The gall-bladder becomes very much dilated, and may be felt as a mere superficial swelling, although this is often not possible owing to an enlarged liver from secondary growths, or to ascites. The engorgement of the liver with the bile that cannot escape also leads to an enlargement of that organ. Pressure on the portal vein may lead to thrombosis of the vessel and consequent ascites, which in rare cases has been "chylous" in character; similarly pressure on the underlying vena cava determines œdema of the lower extremities, and enlargement of the spleen follows from compression of the splenic vein. Much more rarely occurs gastroectasis due to obstruction of the pyloric orifice by the encroaching pancreatic tumour; and even more seldom has a left hydronephrosis resulted from pressure on the ureter when the tail of the gland has been the seat of the growth. Lancereaux records a case where the superior mesenteric artery was obliterated by pancreatic cancer, with consequent intestinal hæmorrhage.

Symptoms indicative of impaired digestion are of most inconstant occurrence, and are seldom directly referable to the pancreatic lesion. There may be a slight gastric dyspepsia, or this may have been considerable for some years. Some nausea and vomiting are,

on the whole, frequent, the latter sometimes very severe. The appetite is capricious, the tongue is generally clean, and an increase of the salivary secretion has been said to occur. The bowels are irregular, and diarrhœa or constipation may be marked. The stools, which are colourless from lack of bile, seldom contain appreciable fat, though undigested muscle fibres have been noticed, and blood may now and then be present.

Among the more general phenomena emaciation and cachexia are the most important, being rapidly developed, and, to an extreme degree, making, with the progressive jaundice, a picture of the gravest illness. Diabetes is, on the whole, rare, as it is seldom the gland is entirely involved in the morbid change. Slight albuminuria is frequently noticed, and also a liability to hæmorrhage from the mucous membranes of the gums, nose, and bowel, as well as cutaneous petechiæ.

The disease progresses to a fatal issue within a few months, seldom a year after jaundice is established, and it may be but a few weeks. Death is due to the wasting and exhaustion, perhaps accelerated by hæmorrhage, from ulceration of the cancer into bowels or stomach, or sometimes from pulmonary embolism. Should the growth invade the liver, stomach, or other organs, further symptoms referable to such extension may supervene and mask those of the pancreatic affection, and the biliary obstruction may determine an infective cholangitis, marked by the occurrence of ague-like attacks.

Diagnosis.—Although in any given case the presence of cancer of the pancreas may be strongly suspected, it is difficult to be sure that this organ is alone involved, or that it is the primary seat of the mischief. The following quotation from Mr. Mayo Robson's article in the *Encyclopædia Medica* (vol. ix.) undoubtedly expresses the general rule, and rightly emphasises the great importance of persistent jaundice in the formation of a diagnosis:—"Persistent jaundice without pain, associated with tumour of the gall-bladder, is usually dependent on cancer of the head of the pancreas, and, in fact, whenever after forty there is chronic jaundice, associated with a distended gall-bladder, even with pain resembling gall-stone attacks, a suspicion of pancreatic cancer should be entertained." A similar condition at an earlier age might suggest that the growth was of the sarcomatous rather than of the carcinomatous type.

As serving to distinguish malignant growth from other affections of the organ of a more or less chronic character, and associated with very similar symptoms, such, for instance, as pancreatic calculi,

or chronic pancreatitis, the state of the blood may afford useful information. In cancer a very considerable leucocytosis may be observed, which is not the case in the other states. The infrequency also of diabetes and of fatty stools in the cancerous disease as compared with what obtains in the non-malignant affections gives some assistance in arriving at a conclusion, whilst a palpable tumour is much less likely to be detected in the latter maladies.

The grounds upon which a growth when recognised may be located in the pancreas rather than in one of the adjacent organs may be thus summarised. When the pylorus is the seat of a new growth the tumour, if palpable, is more superficial and more movable than a pancreatic neoplasm, and although dilatation of the stomach is more likely to ensue, jaundice and glycosuria are much less probable. Similarly, a greater superficiality and mobility, with freedom from jaundice, characterise a growth of the transverse colon, the condition of which may be the easier ascertained by artificial distension with air. It is practically impossible to discriminate between a pancreatic tumour and one starting in the duodenum or in the common bile duct, for in all a persistent jaundice is an equally marked feature.

Treatment.—Little can be done beyond relief of symptoms by anodynes, carminatives, or aperients, and even this very ineffectually. Removal of the growth after an exploratory laparotomy is not justifiable unless it be strictly localised and quite small. Mr. Mayo Robson advises cholecystotomy in doubtful cases when the condition may be really one of chronic pancreatitis, but in certain cancer of the organ he regards operative interference as "useless and harmful."

PANCREATIC CALCULI

Symptomatology.—The characters and associations of pancreatic calculi have been previously described (p. 367). In many cases their existence is unsuspected, and only revealed post-mortem; and even when symptoms are present, their dependence on concretions is rarely diagnosed with any certainty. Paroxysmal attacks of epigastric pain resembling those of biliary colic, but referred to the mid- and left scapular region, rather than the right shoulder, and with tenderness that is experienced more in the middle line than occurs with gall-stones, are the symptoms that

may suggest the passage of concretions in the pancreatic ducts. At first there is no jaundice, though this may develop later if the common orifice be occluded by the stone. If to these phenomena there be superadded glycosuria, stools laden with fat and containing much undigested meat fibre, or very exceptionally lipuria, then the diagnosis may be made with fair probability, and may even be confirmed by the detection of calculi in the evacuations. When the calculus is of some size it may give rise to a palpable tumour, though this is very unusual. And when associated with inflammation of the ducts, rigors may occur, and the patient's condition resemble that due to infective cholangitis. The passage of a stone may be marked by rigors, hiccough, vomiting, cold sweats and collapse when the pain is severest, very much as in attacks of biliary colic.

Treatment.—The uncertainty of the diagnosis renders operative procedure of doubtful recommendation, though an exploratory laparotomy may be justifiable, and removal of a calculus, if found, may be accomplished. The attacks of pain should be treated as in gall-stone colic by morphia subcutaneously, stimulants such as brandy or ether, or by chloroform inhalations. If there be reason to suspect that the condition is permanent, the treatment will be in great measure that for chronic pancreatitis, in which drainage of the gall-bladder is strongly advocated by Mr. Mayo Robson. Constipation is in all cases to be avoided by the administration of regular aperients. The increased flow of pancreatic juice which follows on the subcutaneous injection of nitrate of pilocarpin has suggested the use of this drug in cases of calculi, and it is said with benefit.

W. H. Allchin.

DISEASES OF THE LIVER

The Normal Anatomy and Physiology of the Liver

The liver occupies the right upper part of the abdominal cavity immediately beneath the diaphragm, to which it accurately corresponds. When *in situ* it is of an irregular prismatic shape, but becomes flatter when removed from the body, the greater part of the organ being situated in the right hypochondrium, extending also across the epigastric into the left hypochondriac region. For clinical purposes it may be noted that it is divided by a peritoneal reflexion (falciform ligament), which extends from before back between the diaphragm and upper surface, into right and left lobes—the former constituting about four-fifths of the whole organ ; on the under surface the separation of these lobes is indicated by a fissure, in the posterior part of which lies the remains of the ductus venosus, and in the anterior part, which continues to a notch on the anterior border, is found the obliterated umbilical vein. The upper, anterior, and outer or right surfaces, which pass into one another by rounded borders, lie in contact with the diaphragm and wall of the abdomen, the highest point on the right side being at the level of the line between the eighth and ninth dorsal vertebræ, that on the left side being nearly a vertebra lower. The left limit of the left lobe is usually in the vertical plane drawn through the tip of the ninth costal cartilage (left lateral line of Addison, see p. 6). From this the sharp anterior edge may be traced obliquely down to the right, emerging from under the left costal arch about the tip of the eighth cartilage, and thence across the epigastrium to the right costal arch at the tip of the ninth cartilage, where it follows just within the costal margin round into the mid-axillary line, where its lowest point is at the level of the lower border of the tenth rib. From this it ascends along the line of the eleventh and twelfth ribs towards the spine, which it reaches at about the second lumbar vertebra. The posterior surface of the viscus, like the anterior, superior, and outer, is in contact with the parietes, lying against the diaphragm across the back of the abdomen. For a space of 3 to 4 inches by $1\frac{1}{2}$ to $2\frac{1}{2}$ inches at the right end this surface is uncovered by peritoneum, being separated from the diaphragm only by connective tissue, in which free anastomoses occur between the portal and systemic vessels. Lying behind the left extremity of this area is the right suprarenal body, and to the left of this is the inferior vena cava in a fossa or even canal in the liver substance. The Spigelian or caudate lobe is next to the left, between which and the posterior portion

of the left lobe is the fissure of the ductus venosus. The œsophagus grooves the back of the left lobe, close to the caudate.

The lower surface of the liver is in contact with the following viscera, which more or less distinctly indent it: the upper surface of the stomach, and the lesser omentum in connection with the left lobe, the pylorus, duodenum, hepatic flexure, and transverse colon, the upper half to two-thirds of right kidney behind the colic impressions, which extend forwards to the anterior border. Occupying about the centre of this surface is the transverse or portal fissure, at which the portal vein, hepatic artery, and duct enter the liver, and to the edges of which the two layers of the lesser omentum are fixed. In front of this fissure is the quadrate lobe, against which the pylorus lies, and to the left of the quadrate is the gall-bladder, the fundus of which generally protrudes slightly beyond the anterior margin of the organ which is notched.

The surface relations of the liver are subject to considerable variation determined by age, degree of distension of the stomach and intestines, movements of respiration, shape of the thorax, tight lacing, besides such as are due to congenital displacements or to disease. Except where the anterior surface crosses the epigastric region, about midway between the ensiform cartilage and umbilicus, the organ is under cover of the ribs, scarcely extending below the right costal margin. At a point in this border just outside the edge of the rectus muscle, and immediately internal to the ninth costal cartilage, the fundus of the gall-bladder projects. The summit of the right lobe is on a level just below the nipple, almost corresponding to the level of the fifth chondro-costal articulation. The summit of the left lobe is somewhat lower, being on the level of the sixth chondro-costal articulation, nearly an inch below the nipple on that side. A line joining these points would roughly correspond to the upper limit of the liver, and would pass immediately above the sterno-xiphoid articulation. The extreme left limit of the organ is about $1\frac{1}{2}$ to 2 inches to left of sternum (left lateral line). The greatest vertical measurement of the organ is 5 to 7 inches close to the right surface; the greatest transverse extent is about 8 or 9 inches; the greatest antero-posterior measurement above the right kidney is 4 to 6 inches. The average weight in the male adult is 50 to 60 ounces, and in proportion to the weight of the body ranges from birth to maturity from $\frac{1}{18}$ to $\frac{1}{36}$.

Minute Structure of the Liver.—The liver essentially consists of a mass of cells of about $\frac{1}{800}$ inch in diameter, and of a polyhedral shape from compression; no definite cell wall is to be seen. They are granular, the protoplasm of which they are composed containing a variable amount of fat particles and globules; and the nuclei are distinct and round. These cells are arranged in polygonal masses—lobules—$\frac{1}{12}$ to $\frac{1}{25}$ inch in section, which are separated more or less com-

pletely from each other by a small amount of connective tissue which is continuous throughout the organ, and also with the somewhat denser layer that forms a delicate capsule to the gland immediately beneath the peritoneal coat. Prolongations of this same areolar tissue pass into the transverse fissure, and form a sheath for the subdivisions of the portal vein, hepatic artery, and bile ducts, enclosing them in what are known as the portal canals. Similar investments surround the branches of the hepatic vein—the hepatic canals. Lymphatic vessels, which "originate from the lymphatic spaces around the capillaries of the lobules" (Quain) run in both sets of canals. The arrangement of the terminal blood-vessels within the lobules is very characteristic. The ultimate branches of the portal vein (interlobular) and of the hepatic artery subdivide at the circumference of the lobules, which they penetrate, forming a close capillary plexus continuous with that of the commencing hepatic vessels which occupy the centre of the lobule (intralobular). These last uniting together and occupying, as said, the hepatic canals, ultimately form the hepatic vein which enters the vena cava. The meshes of the capillary plexus, irregular in shape, are occupied by the hepatic cells, which on section of the lobule appear as if arranged in a radiating manner from the central hepatic vein, and the blood-vessels when injected present a similar appearance. The bile ducts commence by extremely fine canaliculi between adjacent cells, thus forming minute networks throughout the lobule, at the surface of which they join up to form the bile ducts, and then, passing into the portal canal, gradually increase in size until they finally unite into the hepatic duct; this leaves the liver at the transverse fissure, subsequently joining the cystic duct to form the common bile duct, which opens into the duodenum. The interlobular ducts are lined by a short columnar epithelium disposed on a basement membrane outside of which is the interlobular connective tissue. The larger ducts in the portal canals are also lined by columnar epithelium, and opening into them are "saccular and tubular recesses, which are often branched and anastomosing, and may be beset all over with cæcal projections" (Quain). In the walls of the largest ducts are numerous mucous glands.

The branches from the left vagus and from the solar plexus are distributed to the liver, and have been traced to their termination in the blood-vessels and ducts, and also between the hepatic cells, over which they ramify.

The *gall-bladder*, which is pear-shaped and about 3 inches in length, and of a capacity of 1 to 1½ fluid ounces, is in great part covered by peritoneum, beneath which is a thick fibrous coat containing plain muscular fibres, and lined by a mucous membrane presenting a reticulated appearance, and is covered by a columnar epithelium. The neck, which is somewhat twisted, passes into the cystic duct, its inner surface being thrown into a series of crescentic folds forming a partial

spiral. The length of the cystic duct is 1¼ to 1½ inches, of the hepatic duct slightly less, and of the common bile duct about 3 inches; the average diameter being about ¼ inch.

The Functions of the Liver

These are several and varied. The liver is a secretory gland concerned in the formation of the bile, which is partly excretory in character, and partly an adjuvant to the pancreatic juice in its digestive power. Placed as the organ is in a direct vascular communication with the gastro-intestinal tract by means of the portal circulation, it receives the absorbed proteids, carbohydrates, and salts, which it deals with in a way that fits some for further tissue metabolism, and effects such changes in others as permit of their easy and harmless elimination. Together with these normal products of digestion poisonous substances frequently reach the liver, whether these be taken in among the ingesta as such, or are the result of faulty changes in the alimentary canal; and, further, there is reason to believe that certain micro-organisms, pathogenetic and others, find their way to the liver by the same channels. Many of these injurious agents are arrested in the liver and rendered innocuous. Stored up in its cells also is a large quantity of fat readily available for oxidation, and also an accumulation of the iron resulting from the normal disintegration of the red blood corpuscles. Its main purport, therefore, is connected with nutrition both in its anabolic and catabolic aspect, incidentally subserving the functions of digestion and excretion, as well as being possessed of antitoxic and bactericidal powers.

In considering the activities of the liver, it is necessary to remember that besides the true hepatic cells which form the lobules there is an epithelial lining to the biliary passages, and both groups of protoplasmic elements are more or less involved in the functional output of the organ.

The bile.—About 1 to 1½ pints of this fluid are daily formed in the human subject, being continuously secreted and stored in the gall-bladder, whence it passes into the duodenum during digestion, being stimulated thereto by the flow of the acid chyme over the biliary papilla. It varies in character and composition according as it is obtained from the gall-bladder or from a fistulous opening in the hepatic duct. In the latter case it is a thin, diffluent fluid of a sp. gr. of 1010, but in the gall-bladder it becomes viscid and concentrated from mixture with mucin, having then a sp. gr. of 1030 to 1040. Alkaline in reaction and intensely bitter, it varies in colour, either golden yellow, reddish brown, or green. The following table shows the average percentage of composition from the two sources:—

	Gall-bladder.	Hepatic ducts.
Water	86	98.2
Solids	14	1.8
Bile salts	10	.76
Cholesterin, lecithin, fats, soaps	.6	.12
Mucin and pigments	2.7	.15
Inorganic salts	.7	.75

From this it will be seen that the bile contains no enzymes.

The *bile salts* are sodium compounds of glycocholic and taurocholic acids, the former being the more abundant. Both these acids consist of chololic or cholic acid combined with glycocoll or glycin and taurin respectively, and are secreted by the hepatic cells. The glycin and taurin (amido-acids), the latter of which contains sulphur, are derived from the proteids of the tissues and of the food. The special functions of these salts are to act as solvents of the cholesterin and to assist in the absorption of fats. Only a very small quantity of these is excreted into the fæces, the greater part being absorbed from the bowel. They are active excitants of the flow of bile.

The usual test (Pettenkofer's) for these acids or their salts is the production of a purple colour on gently warming with strong H_2SO_4 and cane sugar. Other substances, however, give a similar colour under like conditions.

The *pigments* of the bile, bilirubin and biliverdin, are derivatives of hæmoglobin, and are formed by the liver cells. The hæmatoidin of old blood clots is identical with bilirubin. By oxidation with nitric acid the reddish brown bilirubin becomes converted into the green biliverdin, subsequently changing to blue, red, and finally yellow (Gmelin's test). These pigments readily combine with alkalies, forming compounds soluble in water, and with alkaline earths which are insoluble. Most gall-stones consist of a combination of calcium and pigment.

Cholesterin and lecithin are products of the disintegration of nervous tissue, and like the pigments are excretory substances.

The greater part of the iron derived from the hæmatin moiety of the hæmoglobin is stored in the liver, a trace only being excreted in the bile.

The secretion of bile is regulated chiefly by the rate and quantity of the blood flow, and is apparently uncontrolled by the nervous system, except so far as this governs the vessels. It takes place at a low pressure, though this is higher than the blood pressure in the portal vein.

Reference has been previously made to the use of the bile as a stimulant to intestinal peristalsis, and as aiding the pancreatic juice in the process of digestion (p. 21).

GLYCOGENIC FUNCTION.—This has been already considered in

connection with the subject of diabetes (Vol. II. p. 179). It suffices, therefore, to say that, notwithstanding the great fluctuations in the intake of carbohydrates in various forms, the quantity of dextrose remains almost constant (under .2 per cent) in the blood, and that this uniformity is maintained in great part by the liver, to which the digested and absorbed carbohydrates are taken as dextrose and then stored up in the liver cells as glycogen, thence to be reconverted into dextrose, and thrown into the systemic circulation as required for combustion in the tissues, especially the muscular. Hence the glycogen may be regarded as an internal secretion of the liver cells. By the withdrawal of carbohydrates from the food or by muscular exercise the formation of glycogen is diminished; the necessary dextrose is then formed by the liver from the digested proteids. This dealing with the proteids by the liver results in the *formation of urea*, which to a large extent takes place normally, any excess of proteid over that which is required by the living cells of the body being so treated; whilst the leucin and tyrosin which result from pancreatic digestion are also converted into urea in their passage through the liver. And it is further probable that some of the products of muscular metabolism are also changed into the same substance by the liver. It is in the behaviour of the hepatic cells towards the proteid and carbohydrate ingesta, and to a certain extent the fats which they temporarily store up, that the most important functions of the liver are to be found.

General Etiology of Hepatic Disease

The intimate relationship which exists between the liver and the alimentary canal through the portal system, and the enormous and varying blood capacity of the organ, are important circumstances in the etiology of hepatic diseases. The former furnishes channels of invasion for noxious agents, toxic or bacterial, derived from the gastro-intestinal tract into which they have been introduced, or in which they have been formed; the latter predisposes to the establishment of an excessive congestion of the viscus with the morbid changes consequent thereon. The direct continuity of the bile ducts with the lumen of the duodenum admits of the entrance into the biliary passages of parasites, both macro- and microscopic, from the bowel, or an extension of catarrhal states from one to the other. The anatomical arrangement of the opening of the bile duct explains the facility with which it may be occluded, thus preventing the escape of the bile.

The several conditions which may be regarded as exercising a predisposing influence in the production of hepatic disease are—

Age.—No age is exempt. At birth the liver may be found affected as the result of congenital syphilis; and jaundice in the new born is a well-recognised malady. A form of cirrhosis is not unknown in children, and an enlarged liver from congestion due to heart disease may occur in them, as at any age. Even new growths have been met with in early life, though, like malignant disease elsewhere, they are commoner after middle age.

It is doubtful how far *sex* of itself confers any liability, except so far as it favours the incidence of any of the more direct causes of hepatic disease; though the much more frequent occurrence of gall-stones in women is not altogether explained by their habits of life. Whilst statistics show that primary cancer of the liver is common in men, secondary growths are oftener met with in women, due, no doubt, to the frequency of mammary, uterine, and ovarian cancer as primary affections.

Heredity.—Although often claimed as a predisposing factor, its influence is very doubtful. There is no reason to believe that it exerts other than the most indirect effect in the production of malignant disease of the organ, most cases of which are secondary to growths elsewhere, and in the primary affection only a very small percentage give a history of a similar affection in their family. Attention has recently been drawn to the occurrence in parent and child, in one case even in six individuals over three generations, of attacks of recurrent jaundice associated with hepatic cirrhosis and splenomegaly (see *Trans. Clin. Soc.* 1902, p. 155).

Closely connected with the idea that prevails so exclusively of attributing to the liver many disorders that are comprised under the vague term "biliousness" is the assumption that these maladies are often the result of family or hereditary tendencies, which, no doubt, in the great majority of cases, are really due to a similarity of habits and conditions of life among the several members of the family, even in successive generations. At the same time, having regard to the relation of the liver to tissue nutrition and metabolism, it might be expected that perversion of these functions might be transmitted from parent to offspring, since it is among maladies of this class that the most definite examples of transmission are to be found. And it is in the writer's opinion probable that some of the obscurer phenomena of malnutrition often included within the expressions "gout" and "gouty" are manifested in families as the result of hereditarily descended structural defects — whether chemical or molecular—in which the liver is largely concerned.

Worry and anxiety have been frequently noticed as precursors

to the development of malignant disease of the liver, but whether this is more than coincidence is doubtful, and such effect as they may have is by lowering the body resistance.

Among the most efficient and the most frequent of the exciting causes of hepatic disease are *habits of life*, and of these errors in diet stand first. Over-indulgence in eating, especially rich, fatty, and nitrogenous food, loads the liver with more material than it can satisfactorily deal with, and thus leads to functional impairment, and in time to gross structural change. This is well seen in the case of alcohol, which is directly responsible for such morbid conditions as cirrhosis and fatty liver. Great differences, however, exist among individuals as to the readiness with which these changes are induced, some being remarkably tolerant, whilst others more speedily succumb. Much depends, no doubt, upon the amount and the form in which the alcohol is presented, whether wine, beer, or spirits, and also upon the quantity of food taken; so long as this is sufficient the ill effects on the liver of the alcoholic excess frequently remain to a great extent in abeyance, becoming pronounced when the appetite fails. If to over-eating be added deficient exercise, the probability of hepatic trouble is increased, due to the establishment of congestion of the liver, which the conditions of the portal circulation especially favour.

Although generally capable of dealing satisfactorily with the toxic substances which reach the organ by the portal blood, it sometimes happens, as, *e.g.*, in the case of phosphorus, that this is not so, and the liver itself undergoes structural damage in consequence.

Climatic states also exert an influence in the production of hepatic disease, especially hot and damp regions in the tropics, and this independently of dietetic errors and a sedentary life, which are not infrequently associated with the prejudicial habitat. An active hyperæmia of the organ is the morbid state set up, at first of an acute character, which becomes chronic as the exposure to the injurious environment continues.

Trauma.—The liver, though well protected by the ribs, is nevertheless, from its size, subject to injury of various kinds. Sudden blows or falls may rupture the organ, or it may be wounded from gun-shots, stabs, and the like.

Whether *secondary to disease of other organs*, or involved by extension from adjacent structures, or as sharing in a general malady, the liver is very frequently affected. Reference has been made to the relationship of the viscus to the gastro-intestinal canal, and the facility which this offers to the setting up of morbid changes in the

liver, whether by the biliary passages or by the blood-supply. In the febrile state the functions of the organ concerned with the elaboration of the products of digestion and metabolism would seem particularly to suffer.

W. H. Allchin.

The Morbid Anatomy and Histology of the Liver and Bile-passages

Owing to the large size of the liver and the important position in which it stands with regard to the entire alimentary tract, the number of morbid conditions which affect it and the bile passages is considerable. In the majority of cases, too, the hepatic changes are not isolated, but are accompanied by other changes in the body, whether they are the causes, or the results, or merely the accompaniments of those changes.

(1) **Simple vascular changes**—(*a*) *Chronic venous congestion.*—The alteration which is produced in the liver under these circumstances is very characteristic. The liver itself is enlarged and hard, its capsule is smooth, and its edge more rounded than normal. Its colour varies from a deep purple, which occurs when the condition of venous congestion has not been of long duration, to a mottled colour, in which red, yellow, white, and brown are to be recognised in varying proportions in different places. The latter appearances are characteristic of a long-continued venous congestion, and have caused the organ to be called the *nutmeg* liver.

Histologically, the central vein of the lobule is found to be widely distended with blood, and the capillaries throughout the lobule, but more particularly those in the immediate neighbourhood of the central vein, are dilated and engorged. These changes induce a variable, but generally considerable, degree of pressure atrophy of the liver cells, which in its turn is most manifest in the centre of the lobule. The red foci in a nutmeg liver are therefore situated chiefly in the centres of the lobules.

The congestion which is present throughout the liver affects the red blood corpuscles themselves. Many of them disintegrate, and the pigment which is formed as the result of their destruction is stored up, in large part, in the hepatic cells closest to the seat of its formation. These same cells are, moreover, undergoing a fatty degeneration in the course of their atrophy from pressure, and hence around the red central focus there is a somewhat irregularly

disposed zone which is more pronouncedly white or brown according as more fat or more pigment enters into its composition.

The bright canary yellow foci or lines that are visible in a nutmeg liver are situated in the periphery of the lobule. Microscopically it may be found that the bile pigment has only stained the lining epithelium of the bile ducts themselves, or that the colour has diffused through the entire wall of the bile duct, and has passed into and stained the peripheral hepatic cells of the lobule.

In addition to these changes the nutmeg liver frequently shows a slight excess in the amount of its fibrous tissue.

(*b*) *Infarction.*—Infarcts of the liver are very rare compared to their occurrence in other organs, and the conditions which underlie their formation seem to be different. For it may almost be said that heart disease is never the cause of infarction of the liver, which is associated most commonly either with malignant disease of the organ, or with severe injuries, or with pylephlebitis. The infarcts may be of the red or of the white variety, and both kinds may co-exist in the organ. Of the two the red are the commoner, and are usually the smaller; anæmic infarcts of the liver are hardly ever found except of considerable size. Infarcts are generally quadrangular on section and have their bases at the surface, but they are sometimes seen in the very substance of the liver. Microscopically they resemble infarctions elsewhere.

(2) **Degenerations and infiltrations.**—(*a*) *The lardaceous change.*—In the case of this change, which is never present without affecting the liver, the brunt seems to be borne by the capillaries between the individual liver cells, but it is very probable from the appearances that the hepatic cells themselves are also affected. The larger blood-vessels (arterioles and venules, both hepatic and portal) are liable to show the change in their middle coats as in other tissues of the body. The degree to which the change is visible varies considerably in different cases. In one it may be quite slight, in another it may affect the entire lobule, and practically the entire liver. Under any circumstances the change first appears, and is ultimately most marked in the middle zone of the lobule. The usual explanation of this distribution is that the middle zone of the lobule is that which is the especial region of the capillaries terminating the branches of the hepatic artery. The hepatic cells themselves undergo pressure atrophy.

The liver which is affected to any considerable extent by lardaceous disease is much enlarged, its edge is rounded, its consistency is greatly increased, so that the organ feels tough, and it has a

peculiar semi-translucency on section. If a very thin slice of the organ be held up to the light, the situation of the lardaceous material is easily determined by the greater translucency, and then it is seen that the masses are circular or oval or irregular (but with rounded angles), and that there is a narrow line of opaque tissue between two adjacent masses of the lardaceous material. This appearance of a thin slice is not seen in the case of any other change.

(*b*) *Fatty changes.*—Either a true infiltration may be present or

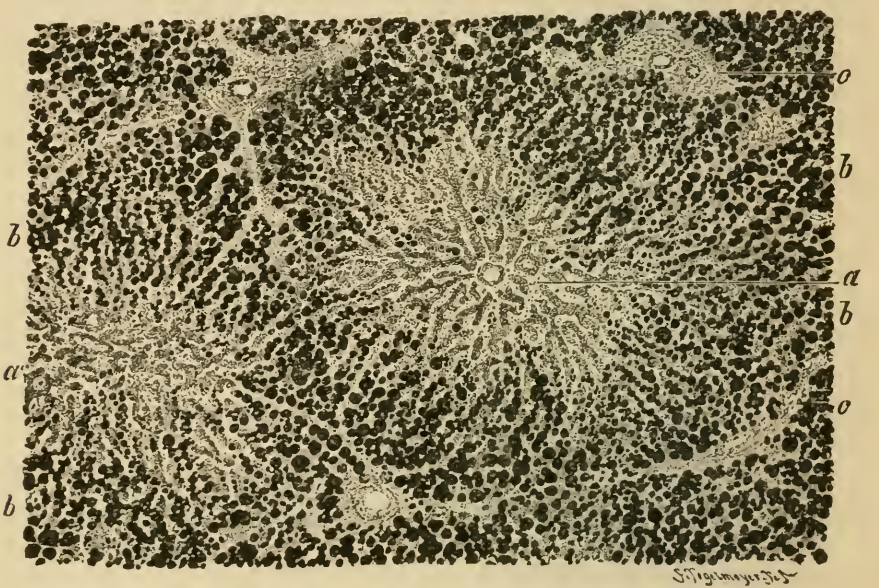

FIG. 12.—Fatty liver (osmic acid and saffronin. × 30). *a*, Central portions of the lobules; *b*, peripheral fatty zone; *c*, interlobular connective tissue. (Ziegler.)

a true degeneration. More commonly, when the change is a degeneration, this is combined with a certain amount of infiltration. The difficulties in deciding which of these two forms of fatty change is present are great, but in the rare instances in which the question is beyond doubt (*i.e.* conditions in which the change is met with at its very commencement) it is found that there is a difference between the portions of the lobule that are affected in the two cases. In very early fatty infiltration of the liver the fat globules are arranged in the periphery of the lobule, that is, in the region of the branches of the portal vein by which it is assumed that the fat is carried to the liver. In an early case of fatty degeneration, on the other hand, such as that which results from poisoning by phosphorus,

arsenic, or antimony, the fatty change is seen to affect the entire lobule.

A fatty liver is generally enlarged, and its edge is rounded. Its aspect is pale or yellowish, and on section the surface has a greasy look. In very advanced cases droplets of fat may be seen on the surface of the knife that was used to make the section. Owing to the amount of fat present the consistency of the organ is diminished, and it readily breaks under the pressure of the finger. The total amount of true liver substance present is diminished proportionately to the amount of fat. This can be easily seen in specimens which have been kept in alcohol, and particularly in those which have passed through the stages necessary for embedding in paraffin. In sections prepared after this manner, the former situations of the fat drops are marked by a number of circular lacunæ, and the amount of true liver substance that is left is often surprisingly small.

(*c*) *Siderosis* is a condition in which iron is deposited in the cells of an organ. It is principally seen in the case of the liver, and the disease in which it occurs to the most marked extent is pernicious anæmia. The deposition of iron takes place in the hepatic cells of the middle and outer zones of the lobule, and is so regular that when a microscopic section has been appropriately treated, the position of the iron forms a delicate tracery over the section that can readily be observed by holding the specimen up to the light.

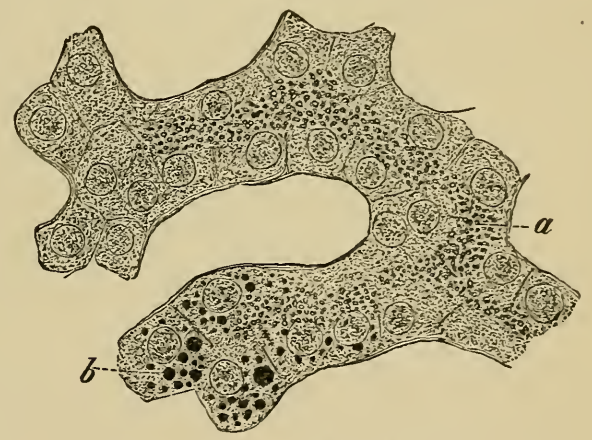

FIG. 13.—Siderosis (perosmic acid and carmine. × 250). *a*, Pigment granules; *b*, cells undergoing fatty degeneration. (Ziegler.)

(*d*) *Vacuolation.*—Of this change in the liver cells, which is probably degenerative and not uncommon, but little is known. The change, indeed, is not confined to the liver, although it is more commonly seen in this organ than elsewhere. The vacuoles are of various sizes, but in some cases almost reach to the limits of the cell and push the nucleus on one side. More commonly two or three vacuoles are present, and then the nucleus is, as it were, suspended in the centre of the cell. The vacuoles themselves are

filled with a colourless fluid, and the change is one which accompanies the more severe degenerations and affections of the liver.

(*e*) *Focal necrosis.*—In several of the acute infective diseases there is a tendency for portions of the hepatic tissue to undergo a definite necrosis. This is particularly the case in typhoid fever. The foci of necrosis are generally situated around small branches of the portal vein, and the destruction of the tissue is so complete that nothing is visible except a granular *débris*, which stains badly and gives only the slightest evidence that it was formerly composed of cells. The foci are commonly rounded in shape, and there is a very considerable regularity in their size. As the result of the degeneration the foci are somewhat softened, but there is no formation of small softened spots in the liver which could lead it to be supposed from the macroscopic appearances that the organ was the subject of focal necroses. The foci are sometimes the seat of accumulations of bacteria. Although typhoid fever is the commonest condition in which these lesions are seen, their existence is not confined to that disease. They have been met with in ulcerative colitis, in eclampsia, and it is probable that they exist in a large number of acute infections of the intestinal tract.

(*f*) *Acute yellow atrophy.*—Macroscopically the condition of the liver in acute yellow atrophy is one of intense disorganisation. The organ is much smaller than normal, weighing, perhaps, two pounds instead of over three, is extremely flabby, and shows on section that it is composed of two kinds of material, one of which is red, the other yellow. The gall-bladder is generally empty, but it may contain a small amount of pale bile-stained fluid.

The histological changes are of so profound a kind that it is difficult to believe that the section under the microscope consists of hepatic tissue at all. The appearances differ somewhat in the yellow and in the red patches. In the reddish patches the hepatic disorganisation is complete. The cells are broken up into *débris* which hardly stains at all or shows the presence of definite structural elements. A small number of leucocytes may be present, and these stain more or less normally, but this is all. The presence of numbers of red blood corpuscles both indicates the fact that hæmorrhages have occurred and explains the red colour of the patches. In the yellow portions, although to the naked eye they appear to be the more profoundly altered, the changes are not so advanced, and the fact that one is looking at liver substance is still recognisable. But the cells have become granular, and in many places they have broken down altogether. At the same time there is present in these patches a

considerable increase in the amount of fat, but the globules are no longer contained within cells, but lie free. In the red patches the same large numbers of free fat globules are not found as in the yellow, nevertheless some are sure to be present. In addition to the changes which have been described, bacteria of different sorts will be seen in both varieties of degenerated material; but it is impossible to say how far they are concerned in the production of the

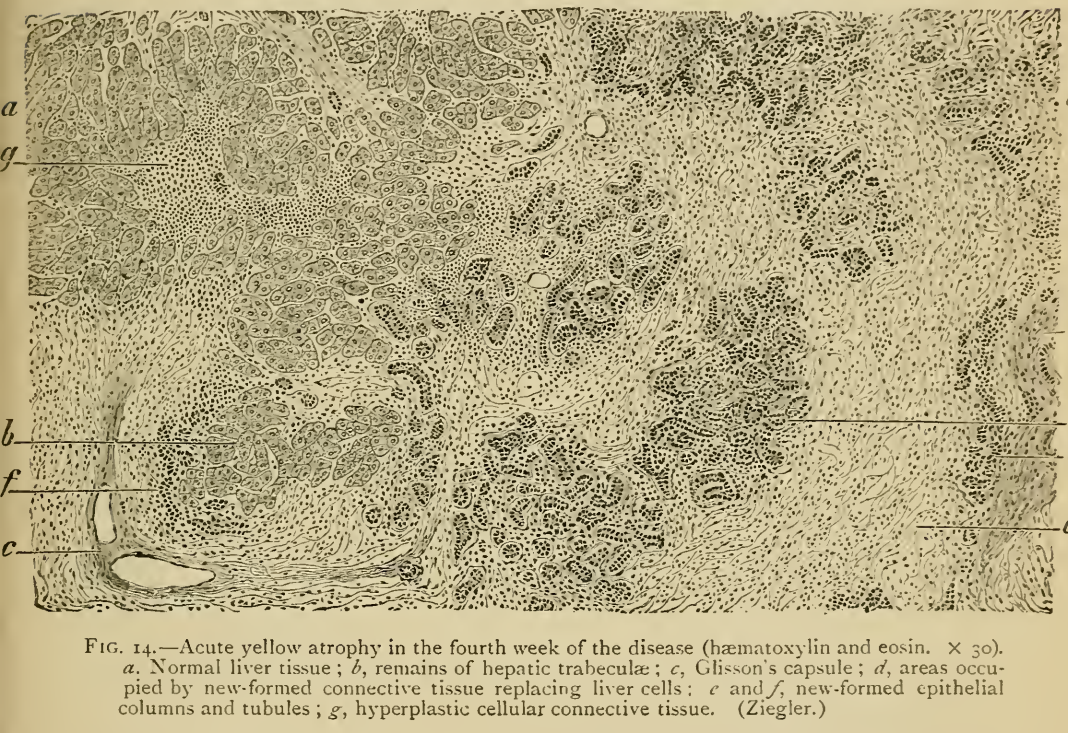

FIG. 14.—Acute yellow atrophy in the fourth week of the disease (hæmatoxylin and eosin. × 30). *a*. Normal liver tissue; *b*, remains of hepatic trabeculæ; *c*, Glisson's capsule; *d*, areas occupied by new-formed connective tissue replacing liver cells; *e* and *f*, new-formed epithelial columns and tubules; *g*, hyperplastic cellular connective tissue. (Ziegler.)

process, or how far they are subsequent and, so to speak, accidental invasions of the degenerated hepatic material from the intestine.

If the disease have been some weeks in existence, and have run a somewhat more chronic course than is normal, there may be found in the less altered portions of the liver a proliferation of the connective tissue elements of the organ with the production of a definite amount of new fibrous tissue. In the midst of this latter are numbers of cells arranged in strands bearing a close resemblance to those appearances which in certain other diseases of the liver are supposed to represent a new formation of bile ducts. Such an appearance is not likely to be seen unless the disease have lasted at least four weeks, or about double its usual length.

It is a question whether acute yellow atrophy should not be included amongst the true inflammations of the liver. It is in the highest degree probable that the disease is infective, in the sense that it depends upon the action of micro-organisms and their toxins, and from this point of view it is certainly inflammatory. Nor does the fact that degenerative processes are so much in the ascendant modify the question, for it is well known that the character of an inflammation differs greatly in different cases, and that when the tissue affected is highly cellular and the irritant is of great intensity, the necrotic phases of the process are apt to be overwhelming. The same arguments are applicable in the case of focal necrosis.

A condition very similar to that obtaining in acute progressive atrophy is seen in cases of acute phosphorus poisoning. In chronic phosphorus poisoning there occurs a pronounced fatty degeneration of the liver of a more or less ordinary type, but when the poisoning is acute it is impossible to determine from the appearances of the liver alone whether phosphorus poisoning or acute yellow atrophy has led to the patient's death.

Leuchæmia.—A condition affecting the liver which may be mentioned here, although it hardly comes into the category of an infiltration or a degeneration, is that which occurs in leuchæmia. In this disease there is generally found a considerable increase in the number of leucocytes that exist in the liver, principally in the neighbourhood of the portal venules, so that the acini appear to be surrounded by a zone of grayish material. At the same time the general increase in the number of leucocytes throughout the organ causes it to be much paler than normal. Sometimes definite masses of cells are present and form macroscopic tumours similar to those which occur in lymphadenoma.

Microscopically the leucocytes are seen to be chiefly aggregated in the connective tissue at the periphery of the lobule, but numbers are also present in the capillaries between the individual liver cells, and in any blood-vessel that may be seen in cross section they are sure to be present, as elsewhere in the circulation, in greatly increased numbers. Special staining will sometimes show that the great number of leucocytes present are of the oxyphil granular type ; in other cases they appear to be lymphocytes.

(3) **Inflammatory affections.**—Truly inflammatory affections of the liver are not of very common occurrence. They are practically confined to the conditions of acute hepatitis and the various changes in the organ which are accompanied by suppuration.

(*a*) *Acute hepatitis.*—Acute hepatitis is an uncommon condition judging by the comparative rarity with which are seen those histological alterations that, elsewhere, are regarded as inflammatory. Nevertheless, cases in which the connective tissue of the organ is the seat of a marked small cell infiltration are occasionally met with. Mostly, the condition is seen in company with a general increase of fibrous tissue such as occurs in the chronic fibroses of the liver. Macroscopically there is generally nothing to indicate that the organ is the seat of inflammation. It is unaccompanied by congestion, so far as can be seen, and swelling is absent.

A condition which is variously termed "hepatitis," "congestion," "acute determination of blood," etc., occurs with considerable frequency among Europeans resident in the tropics. As to the exact nature of the changes concerned in producing this affection we are quite ignorant, but there is no doubt that the liver is enlarged more or less temporarily and is acutely tender. How far the condition is due to dietetic, or alcoholic, or malarial causes it is difficult to say (see pp. 391-426).

(*b*) *Pylephlebitis.*—In this condition there is a more or less diffuse suppuration in the liver confined to the portal vessels and their neighbourhood. It commences by phlebitis with thrombosis of the portal vessels, which is induced by the lodgment of some infective material from the intestinal area. Once the inflammation has commenced, it advances by continuity of thrombosis and of the vessel walls and the connective tissue in which they lie, until the whole or the greater part of the portal system is affected. The disease does not generally lead to any marked changes of the surface of the organ, but when an incision is made into it, it is at once seen that the tissue is intersected in all directions by dilated pus-containing cavities, which at certain points form definite abscesses with very ragged outlines and walls. As a rule the pus in the cavities is creamy and yellow, but sometimes it is grumous from the admixture of broken-down blood-clot.

In a great majority of cases the cause of the pylephlebitis is some inflammatory affection of the intestines, and perhaps the commonest seat for the primary disease is the appendix.

In pylephlebitis the suppurative condition rarely extends beyond the liver. The reason of this is, that the process in the liver itself is so extensive that it destroys life before time has been given for it to invade other tissues by direct extension. Sometimes, however, large branches of the hepatic vein become involved, and then the process may become generalised by way of the circulation. This

does not, however, make any great difference in the severity of the symptoms (see page 552).

(*c*) *Pyæmic abscesses.* — The liver is sometimes the seat of abscesses that are definitely pyæmic, offering no differences from the suppuration that occurs in joints and in the subcutaneous tissues in pyæmia. For the most part the abscesses are of minute size, but sometimes they are fairly large.

(*d*) *Suppuration around a foreign body.*—Suppuration in the liver induced by the presence of a foreign substance may be met with under numerous conditions, but the commonest is when a hydatid cyst suppurates. In this case the suppuration commences around the cyst at the expense of the liver substance. But it involves the hydatid cyst secondarily, so that when it is opened the fluid that escapes is turbid and it may be creamy. The pus is, of course, mixed with the ordinary contents of a hydatid cyst, but the parasites are all dead. In fortunate cases, after suppuration has occurred, the process recedes and then the fluid portions of the pus are absorbed, with the ultimate production of an encapsuled putty-like mass, in which portions of degenerated hydatid cyst wall are visible and hooklets may be found. The capsule of the mass itself is often calcareous.

(*e*) *Specific inflammations*—i. *Syphilis.*—The liver is one of the organs upon which syphilis exerts a great influence, both in the acquired and in the inherited form of the disease. Changes that are met with in the acquired form consist in the formation of gummata; in the congenital disease gummata may also be present, but far more commonly a special variety of fibrosis of the organ is met with. In a gummatous liver two facts are striking: the first is the presence of a larger or smaller number of foci of cheesy material, and the other is the presence of a large amount of semi-translucent, gray, or pinkish, newly formed fibrous tissue which surrounds the cheesy masses and spreads from them, as centres, throughout the organ, reaching up to the capsule in many places. In an early stage of the condition the amount of fibrous tissue that is present is small, and the amount of cheesy material is large. Still earlier there can be no doubt that the syphilitic disease shows itself by the presence of a newly formed material of the granulomatous type, but evidences of this condition are only found in rare instances and at the margins of the gummatous patches. In a well-defined case the microscope reveals nothing more than the cheesy substance and a fibrous tissue which is for the most part dense and contains few cells, but may, in places, be cellular, and give appearances usually

ascribed to the formation of new bile ducts. At this time the fibrous tissue has not begun to contract to any great extent. At a later stage the appearances are altered by the fact that the connective tissue contracts, and the older the condition the greater the contraction. As a result of the presence of large tracts of fibrous tissue in the centre of the liver which reach up to the surface, the surface of the organ becomes puckered, and definite cicatrices are visible. At the same time the amount of cheesy material undergoes diminution, probably as the result of its compression, until at last it may disappear entirely, and there remains a liver which shows one or more deeply depressed and puckered cicatrices. Such a condition is as characteristic of antecedent syphilis as is the existence of gummata themselves (see also page 405).

ii. *Tuberculosis* of the liver is a decidedly rare condition, excluding the cases in which it is affected, along with the other tissues and organs, in a case of generalised miliary tuberculosis. In very rare instances tuberculosis occurs in the liver in the form of caseous nodules similar to those seen in the spleen. The masses are rarely of larger size than a Spanish nut.

iii. *Actinomycosis* occurs somewhat less often in the liver than it does in the other tissues which it affects in man. It leads to the formation of ragged abscesses, which are traversed generally by a number of trabeculæ composed of the undestroyed pre-existing connective tissue and vessels of the affected portion of the liver. The condition, therefore, has a certain resemblance to pylephlebitis. The abscesses are generally large, and send numerous processes of inflammatory tissue (which has not, as yet, broken down) into the surrounding structure. Microscopically, the appearances are those seen in other cases of actinomycosis (see Vol. I. p. 245). The condition may be surmised from the general appearance of the liver, but a diagnosis cannot be made on these lines alone. It is only when the definite nodules of the fungus itself are proved to be present by the microscope that the nature of the change can be affirmed without doubt. It is probable that, in addition to actinomycosis, the liver is occasionally the seat of other forms of streptothrical infection.

(4) **Chronic fibrosis.**—(*a*) *Multilobular fibrosis or cirrhosis.*—In this variety the fibrous tissue is very irregularly distributed throughout the organ, and by its contraction leads to a great diminution in the size of the liver. On this account the change is often called **atrophic cirrhosis**, the name "cirrhosis" being derived from the yellow colour of the organ due to the presence of

confined bile. At the same time the fibrous tissue in its contraction causes an irregularity of the surface of the organ, and has given rise to the name of "hob-nail liver." Lastly, in accordance with the belief that this variety of cirrhosis is principally caused by over-indulgence in spirits, the change has been termed "alcoholic cirrhosis," or, more specifically, "gin-drinker's liver."

The fibrous tissue in cases of multilobular fibrosis runs in broad bands throughout the entire substance of the organ, and the more definite bands may easily be seen on the surface of a section, interlacing with one another and cutting up the liver substance into a number of irregularly polygonal areas of different, but usually small, sizes. The fibrous tissue itself is grey in colour and semi-translucent, and that which is present in the depth of the organ can easily be seen to be in direct connection with the thickened capsule of the liver itself. The fibrous tissue is especially developed in connection with Glisson's capsule and its prolongations, but it is not confined thereto. For careful examination shows that there is a considerable new formation of fibrous tissue which, though it may have originated in connection with the periphery of a lobule, nevertheless strikes out in directions that have no definite relation to the lobules themselves, but in many instances pass right through the centres of them. The effect of the presence of this large increase in the amount of fibrous tissue in the liver not only leads to a diminution in the size and weight (an atrophic liver of this kind may weigh only two pounds, in spite of the large amount of fibrous tissue present), but it also leads to a considerable increase in the density of the organ. The contraction of the fibrous tissue, too, leads to a compression of the portal and biliary canals, and accounts for the ascites, the yellow colour of the hepatic tissue, and the slight amount of jaundice that are met with in the disease. At the same time the liver cells themselves undergo a pressure atrophy, and the actual amount of liver tissue that is present in an advanced stage of the change is very small. The connection of the fibrous tissue strands in the substance of the organ with the capsule is easily recognisable in a microscopic section, and the explanation of the "hob-nail" appearance becomes clear. For it is seen that the capsule is not equally thickened everywhere, but is fairly thin at points where there is no connection with strands from the interior of the organ, and is proportionately thick at points where such connection obtains. In the process of contraction of the fibrous tissue, therefore, the capsule tends to be drawn in at certain points, while the relative laxity of the capsule at other points allows of the

slight extrusion of those portions of liver tissue which are being compressed on all other sides. This explains the appearances satisfactorily, but it is doubtful whether it is the whole explanation. It has been shown that there is a tendency for a new formation of true hepatic substance in these situations, and it is probable that the protuberances on the surface of a fibrotic liver are not merely due to altered distribution of pre-existing liver substance, but are also due to definite new formation of liver cells.

In the strands of fibrous tissue there are always to be seen collections of cells which are arranged in parallel strings consisting of two lines closely apposed. A single pair of the strings of cells gives an appearance very similar to that of a small portion of a Henle's loop in the kidney. Although there is the greatest divergence between the views of different authors, the tendency at the present day is to believe that the appearances are due to the definite new formation of tubes designed to functionate as bile ducts, and that they are formed at the expense of true liver cells which modify their function for the purpose.

Besides the new formation of fibrous tissue it has been shown (Flexner) that in this variety of cirrhosis there is a considerable new formation of yellow elastic tissue.

As a rule, a striking appearance in the case of a fibrotic liver is the amount of fat that is present. It is difficult to say whether the fat globules which are present in the liver cells are the result of infiltration or of degeneration. There are reasons for and against each view. But there is no doubt that when the amount of fat in the remaining liver substance is great, the actual amount of true liver substance which is left is exceedingly small. For the liver cells undergo a pressure atrophy from both sides, being compressed by the contracting fibrous tissue, and also by the droplets of fat. As a result of the combined change, it may be almost impossible to recognise the tissue microscopically as liver at all.

A special variety occurs in children. It may be met with as early as four or five years of age, but its commonest incidence is between the ages of eight and sixteen. The degree of fibrosis in these cases is such that its equal is very rarely seen amongst adults. In other respects the liver changes are identical in the young and in the old. It is probable that it is a post-syphilitic change of congenital syphilis.

(*b*) *Unilobular fibrosis or cirrhosis.*—Concerning this variety of hepatic fibrosis there is much more doubt than there is as to either

the multilobular or the intercellular forms; and it is far less common than either of the other two.

In its theoretical form unilobular cirrhosis is characterised by the new formation of a fibrous tissue which surrounds the individual lobules. Such a condition, however, is hardly ever seen, although cases are undoubtedly met with in which the adventitious fibrous tissue forms a far closer meshwork than it does in the multilobular type. At the same time it must be allowed that there is a variety of hepatic fibrosis which is associated with an increase in the size of the liver ("hypertrophic cirrhosis") instead of a diminution ("atrophic cirrhosis"), and that the surface of the organ in this variety is fairly smooth, or at most finely granular, and not coarsely granular or "hob-nailed." The increase in the size of the organ in this variety is beyond question, for the liver may weigh five or six pounds. It is, moreover, different from the multilobular variety in colour, being usually of a deep red and not pale or yellow.

On examining a section of such an enlarged liver microscopically, there is seen a very definite fibrosis of the fine-meshed kind that has been referred to above. The actual strands of the fibrous tissue are not very wide, but they are of fairly even width throughout the organ. In this respect they offer a marked contrast to the strands of fibrous tissue in atrophic cirrhosis. It is undoubtedly true, moreover, that the general tendency is for the fibrous tissue to be arranged around the individual lobules. Nevertheless it will be easy to find a spot at which the strand of fibrous tissue cuts right across a lobule, as it so frequently does in the atrophic variety.

In spite of differences, therefore, the unilobular has many resemblances to the multilobular variety, and some authors have maintained that the atrophic is a late stage of the hypertrophic, or, conversely, that the hypertrophic fibrosis ends by becoming atrophic. On the other hand, the majority of authors maintain that the two varieties are completely distinct, not only in appearance, but also in causation. They hold that the unilobular variety is especially bound up with changes that start in the bile ducts, and upon this view speak of the condition as a "biliary" cirrhosis. That this view is a possible one is seen from the fact that the fibrous tissue is particularly well developed in those regions of the liver in which the bile ducts are present, viz. at the periphery of the lobules. Further, there is no doubt that unilobular fibrosis is associated with the presence of a large number of those cellular appearances which are to be regarded as newly formed bile ducts. The fibrous tissue present in cases of unilobular cirrhosis is not so dense, and con-

tains a larger number of nucleated cells than is the case in multilobular cirrhosis. For this reason the condition can more easily be regarded as being of inflammatory origin. The cells themselves are not merely young connective tissue cells, but large numbers of small round cells, presumably leucocytes, are also present. It is, of course, impossible to determine whether these cells are to be regarded as indicating that the condition itself is essentially inflammatory, or whether it simply means that the newly formed fibrous tissue has itself become the subject of inflammation as the result of the action of an irritant which is acting on it from the bile ducts, or the vessels, or the hepatic cells themselves.

So far as the etiology of the multilobular and unilobular forms of cirrhosis is concerned there is still considerable doubt. Numerous experiments have been made to test the influence of irritants, such as alcohol, chloroform, etc., in rabbits, but the results are very conflicting. Adami has found in the "Picton disease" of cattle that a hepatic fibrosis occurs which is directly traceable to invasion of the liver by certain bacteria travelling from the intestine. And other examples of a fibrosis of bacterial origin have been described, particularly in America. In such cases the fibrosis is usually of the unilobular or biliary type, and the assumption is that the bacteria reached the liver by way of the bile passages.

(*c*) *Intercellular fibrosis or cirrhosis.*—This variety of hepatic fibrosis is met with exclusively in stillborn fœtuses and infants that are the subjects of congenital syphilis. It has, therefore, also received the name of *syphilitic* cirrhosis.

The liver in these cases is larger than normal, is generally of a bright yellow colour, and is of a leathery hardness. It shows little or no irregularity of the surface, but nevertheless the amount of fibrous tissue present may be so great that it is recognisable macroscopically. Under the microscope the fibrous tissue is seen to be situated not only at the periphery of the lobules, but also to penetrate into them and lie between the individual liver cells. In many cases the intercellular substance is fully formed fibrous tissue, but in others it is rather fibroblasts which lie between the hepatic cells. In any case the actual picture produced is one which is totally unlike the normal liver. For the liver cells become compressed into irregular polygonal areas of different sizes and shapes, and the lobular arrangement of the liver is lost. At some points the amount of fibrous tissue may be greater than at others, but in the main the distribution is fairly even over the whole liver. Bile ducts and vessels are compressed, and singularly few of them are

visible at all. Such ducts as are present are stained a brilliant yellow by the confined bile, and the liver cells themselves take on the same colour, owing to diffusion of bile pigments into them. This coloration of the liver in congenital syphilitic cirrhosis is not, however, constant.

Sometimes the variety of cirrhosis which has been just described is conjoined with a certain amount of fibrosis of the multilobular type. These cases form a connecting link between the typically

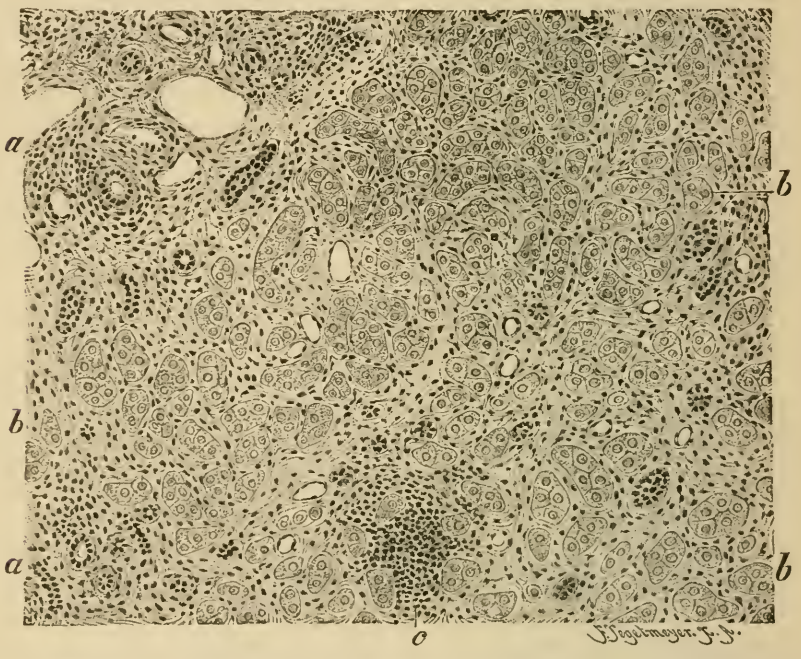

FIG. 15.—Syphilitic cirrhosis (hæmatoxylin and eosin. ×100). *a*, Hyperplastic interlobular connective tissue; *b*, indurated liver tissue traversed by fibrous bands; *c*, clusters of round cells. (Ziegler.)

syphilitic fibrosis and those cases of multilobular fibrosis which are termed post-syphilitic.

(5) **New growths.**—The liver is liable to be the seat of a variety of new growths, but the malignant are more commonly met with than the non-malignant, and secondary growths oftener than primary.

Of *non-malignant growths* only one—the cavernous angeioma—is at all common in the liver. Fibromata have been described, but are rare. Adenomata occur, and are undoubtedly sometimes found in the non-malignant form, but far more commonly they pass over the border between non-malignancy and malignancy, and become definitely carcinomatous.

Cavernous *angeiomata* in the liver are generally of small size, rarely being larger than a walnut. They are situated in the superficial portions of the organ, and their outer surface lies flush with the general surface of the liver. On section they are roughly quadrilateral in shape, having in this respect resemblances to a hæmorrhagic infarct of the liver, and are built up of large blood spaces. Very rarely they may be of large size, and are then of importance from the fact that they may come under the surgeon and lead to fatal hæmorrhage.

Adenomata of the liver occur in normal and in cirrhotic livers as solitary or multiple greyish yellow masses which are usually of small size. The smallest nodules do not seem to be provided with a capsule, but in the larger this is generally well defined. Histologically the growths may consist of a number of convoluted and branched ducts, or of irregular hepatic tissue contained in a more or less voluminous connective tissue. The growth is a very rare one.

Malignant growths of the liver are almost always carcinomatous, and then they may be primary or secondary.

Primary carcinoma of the liver is very rare. It shows itself under four forms. In the first there is only one mass, or at most two or three. This mass is generally found in the right lobe, and is of considerable size; it extends radially. From its appearance it has been termed "massive cancer." In the second variety metastases are formed in the liver itself. These give rise to the "nodular cancer." The third variety is associated with a cirrhotic condition of the liver, and the masses of carcinomatous cells lie between bands of dense fibrous tissue. The organ, generally, has the appearance of an atrophic cirrhosis. This is the "cancerous cirrhosis" or "cancer with cirrhosis." The fourth variety is one which starts in connection with the portal canals and travels along them, sending out offshoots into the hepatic tissue generally. In this variety of hepatic carcinoma definite nodules of a grey colour are usually present, but sometimes the appearances are very similar to those seen in cancer with cirrhosis.

According to the character of the growth the liver affected with primary carcinoma is soft or hard. When the nodules are of relatively large size they often undergo softening in the centre. If the nodule itself be situated on the surface of the organ this softening leads to umbilication of the nodule, a very characteristic feature of carcinomatous nodules in the liver, whether primary or secondary. On the other hand, when the liver is intensely fibrotic the fact that

carcinoma exists is frequently only first recognised when microscopic sections have been examined.

Histologically the primary carcinomata of the liver are almost always composed of spheroidal cells. Sometimes cases are met with that are more of an adenomatous type, and then German authors refer to the condition as *adeno-carcinoma.* The principal difference depends upon the question as to whether the growth have started in connection with the hepatic cells or with the bile ducts. In the latter case it is probably either a typical columnar cell carcinoma or a carcinoma of a transitional type. Confusion must not be made here with carcinoma that starts definitely from one of the larger bile ducts ; this variety of malignant growth affecting the liver primarily will be described later. The bile ducts that are here to be understood are those of microscopic size, and lie close to the bile canaliculi.

In *secondary carcinoma* of the liver the nodules may be so numerous that there is little normal liver substance left, and the size of the organ itself is greatly increased. In other cases only one or two nodules exist, which are, perhaps, no larger than peas. They may be situated in the substance or on the surface of the organ, and in the latter case they generally show marked umbilication, and give a very irregular shape to the surface of the liver. At first the nodules are discrete, but when they have been in existence for some time, and have grown circumferentially, two or more nodules may coalesce at their margins.

Macroscopically the nodules are almost always of a creamy colour, and frequently of a creamy consistency. They seem to be very sharply defined from the liver tissue in which they lie, although they do not appear to be surrounded by a capsule. Macroscopically it is seen that, although the border of the growth is a fairly well defined one, yet the edge is not perfectly regular. Moreover, although there is no formation of a capsule, yet the liver tissue surrounding the nodule shows signs of irritation, and the hepatic cells signs of pressure atrophy. Hence there is a more or less definite zone around the nodule which recalls closely the line of small cell infiltration which lies in advance of the spreading margin of a primary malignant new growth (*e.g.* a carcinoma of the breast).

Secondary nodules of malignant growth in the liver, of course, always possess the characters of the primary growth. Thus there will be a columnar cell carcinoma when the primary growth has been in the intestine, or a squamous cell carcinoma with well-developed cell nests when the primary growth has been in the œsophagus.

Sarcomata far more rarely form secondary growths in the liver than carcinomata, but occasionally they are found, and then they may be composed of any kind of cell which goes to the building of a sarcoma.

Primary sarcoma of the liver is excessively rare, but the changes which it induces are marked. The organ is nodular, greatly enlarged, weighing perhaps ten pounds, and in it are many masses of a soft growth which is often reddish brown from admixture of broken-down extravasated blood. As a rule, the small amount of liver substance which is left is of a brilliant green colour from the contained bile. The microscopic characters are often very complicated.

Cysts.—Amongst the new growths may be included a rare condition, viz. *congenital cystic disease.* The change has certain resemblances with congenital cystic disease of the kidneys, and the two are sometimes found in conjunction. Macroscopically a congenital cystic liver shows, dispersed throughout its substance, a number of small cysts, the number of which differs considerably in different cases, but is rarely very large. Nor are the cysts themselves large, in this respect differing from the cystic disease of the liver which depends upon the formation of adenomatous growths in connection with the bile ducts described later. It is probable that they are, in reality, abnormal bile ducts. Upon this point, however, it is impossible to speak with certainty.

The most important variety of cyst which is found in the liver is that caused by the presence of *hydatids.* A hydatid cyst may be of practically any size, some of the largest containing a pint or more of fluid and being as large as a child's head. They may be single or multiple, but it is commoner for only one cyst to be present. The hydatid cyst itself must be differentiated from the wall of the cavity in which it lies. The former is an integral portion of the parasite, the latter is produced at the expense of the hepatic tissues and forms a veritable capsule. The cyst wall consists of a hyaline material in which are to be seen a number of fine lines lying parallel to one another. In an old cyst calcium salts may be deposited in the wall, or it may have become modified by the extension to it of suppurative processes going on in the liver tissue which bounds it. The capsule in which the cyst lies, on the other hand, consists of more or less fully formed connective tissue which may be comparatively free from cells, or, if it be the seat of a definite irritation, may be infiltrated with exudation fluid and a large number of leucocytes. Although the capsule is dense and smooth on the side towards the parasite, it shades off more or less

imperceptibly into the normal liver tissue externally. The hydatid cyst contains usually a number of daughter cysts, and these, in their turn, may contain other daughter cysts. At the same time it is probable that on some points on the wall of the mother cyst, or on the daughter cysts, or of both, small sessile growths will be found which are ultimately destined to become daughter cysts. In the smaller cysts of all will be found the parasites themselves attached to the cyst wall. Whether parasites are found attached to the wall of the mother and the larger daughter cysts, depends upon circumstances. Sometimes they are found thus attached, but more commonly no parasites are found here, although the cystic fluid contains hooklets that have become detached from parasites that have undergone degeneration (see Vol. II. p. 17).

The Bile Passages

The commonest affection of the biliary passages is the formation of gall-stones. These concretions may be without great effect upon the bile passages themselves, or they may lead to the most profound changes.

Gall-stones.—Speaking broadly, gall-stones are of two kinds, those consisting of cholesterin, and those consisting of bile pigments and lime salts. It is, however, very rare to find that a calculus consists of either of these substances alone; more commonly both are present, though the one or the other is present in preponderating quantity.

Cholesterin gall-stones are of two kinds. Either they are multiple, moderately small, and of fairly equal size, facetted, and covered externally by a thin layer of brown or black material; or they are round or oval, single, faintly stained with yellow bile, semi-translucent, and fairly large. Gall-stones of the first kind constitute by far the greater number of those found; single cholesterin gall-stones, corresponding to the second variety mentioned, are among the rarest of gall-stones. The facetted gall-stones are opaque, but this is due to their outer covering; when they are split, it is seen that the internal portion consists of a colourless crystalline material that cuts easily and has a greasy feel. This material is almost pure cholesterin. The single cholesterin calculus is composed of this cholesterin alone, and has a beautiful crystalline appearance. In the case of both kinds of cholesterin gall-stone the actual nucleus of the calculus is composed of some organic material upon which

the crystals are deposited. The outer covering of the facetted gall-stones consists of calcium carbonate with an admixture of bile pigments.

Gall-stones chiefly consisting of bile pigment have a totally different appearance to those composed of cholesterin. They are always small and multiple, are of an intense jet black colour, and are never facetted, but are always irregular and nodular. They crumble very readily, and are of a non-crystalline structure. They consist principally of a combination of bilirubin and biliverdin with calcium.

The above form the large majority of biliary calculi. In very rare instances calculi are found composed of other substances, *e.g.* calcium carbonate.

The commonest situation in which gall-stones are found is the gall-bladder. Even when they are present elsewhere it is decidedly rare to find the gall-bladder completely free. In a certain number of instances, however, they are met with in the larger bile ducts within the substance of the liver, or it may be in the cystic duct or the common bile duct before or after its junction with the pancreatic duct.

The local effects of gall-stones may be insignificant or severe. The commonest and least significant change which they induce is in the gall-bladder, leading to an alteration in the consistency of the bile which it contains, and rendering it far more ropy, and frequently of a paler colour. The reason of this change lies in the fact that the irritation of the wall of the gall-bladder causes it to excrete a larger amount of nucleo-proteid than normal. This material, which has so close a resemblance to mucus that it has generally been described as being actually that substance, is in cases in which the number of gall-stones is considerable, or in which a gall-stone is impacted in the cystic duct, the only material found in the gall-bladder, the bile pigments being completely absent.

In other cases the irritation caused by the calculi goes farther, and leads to a definite inflammation of the gall-bladder (*cholecystitis*), when a whole series of changes may be induced. The condition may lead to the formation of adhesions between the gall-bladder and the abdominal wall, or there may be formed a large mass of dense adhesions in the portal fissure, or the gall-bladder may become so full and its walls so altered that it ruptures into the peritoneal cavity and produces a general peritonitis; or, again, the inflammation may go on to suppuration, with the formation of an abscess which either ruptures into the peritoneum, or externally, or perhaps into the

intestine. It must not be supposed, however, that all cases of cholecystitis are due to the presence of biliary calculi, for this is not so. In many instances the condition seems to have been induced by the action of an irritant which has reached the gall-bladder by travelling up the bile passages from the intestine.

By far the most important effect of gall-stones is the formation around them of a mass of *carcinoma*. Although the greater number of cases of gall-stones occur without carcinoma of the gall-bladder, it is undoubtedly true that in almost every case of carcinoma affecting the gall-bladder or the bile ducts, gall-stones are, or have been present.

The carcinoma is almost always of the columnar cell type. As a rule the arrangement is fairly typical, but sometimes the epithelium shows a tendency to assume the transitional flattened type which is normally present in the gall-bladder. The growth extends laterally, and invades the liver to a greater or less extent. In an early case it may appear to be only a chronically thickened wall of the bladder until microscopic sections are examined. In other cases the growth forms a mass as large as the fist. Secondary nodules are formed in the lymphatic glands, in the hilum of the liver, and in the liver substance itself, but this is hardly so common as for the primary growth to be the only one.

The BILE DUCTS are not nearly so frequently affected by morbid conditions as the gall-bladder, and even when they are involved the conditions are generally induced in them secondarily. Primary morbid conditions of the bile ducts, in a strict sense, are excessively rare.

The commonest affection concerns the common duct, and consists in the extension to it of an *acute catarrhal inflammation* of the mucous membrane of the intestine. As a result of the inflammation the mucous membrane around the orifice, in the ampulla of Vater, and along the entire length of the duct, becomes red and swollen. It pours out a greater amount of mucus than normal, and the epithelial cells desquamate. As a result of these changes the lumen of the tube becomes blocked. After a few days the condition usually undergoes resolution.

The ampulla of Vater is the occasional seat of a *carcinomatous growth*. The entire mass is rarely larger than a pea, but its situation is such that it is accompanied by the most profound jaundice. Nevertheless the orifice of the duct may not be completely blocked, so far as can be determined by exerting pressure on the gall-bladder at the autopsy. Cases of this description are generally accompanied

by fever and certain other somewhat anomalous symptoms. Secondary growths are rare.

Carcinoma also affects the bile ducts within the liver on rare occasions. Reference is not here made to those cases in which the bile ducts become secondarily affected by extension of a carcinomatous mass that has commenced either in the liver or elsewhere as its primary seat, but to cases in which the bile duct is the primary seat of growth itself. The growth may be a more or less typical columnar cell carcinoma, but sometimes it is an "adenocystoma." That is to say, it consists of a fibrous-tissue stroma with numerous clefts and spaces which are lined by a single layer of cells having the character of endothelial cells, and are frequently dilated to form definite cysts that may be of large size. Growths of this kind may form many metastases in the liver itself having the characters of the primary growth.

Apart from the varieties of obstruction which have been mentioned, a definite *stenosis of the bile ducts* may result from the formation of a mass of connective tissue of inflammatory origin. This condition is rare, and it generally occurs in children that are the subjects of congenital syphilis or have family histories consonant with such an assumption.

In all cases of obstruction a series of effects is to be noticed in the ducts behind the obstruction and in the liver itself. These effects are independent of the actual cause of the obstruction, and consist in a jaundiced condition of the liver substance and dilatation of the bile ducts. The changes in the ducts are accompanied by a thickening of their lining membrane, which may lead to their being mistaken for branches of the portal vein. Microscopically there is a definite increase in the thickness of the wall, and particularly in the amount of fibrous tissue present. The epithelial lining is usually completely wanting.

W. S. Lazarus-Barlow.

General Symptomatology of Hepatic Disease

The wide range of function possessed by the liver determines an equally extensive range of symptoms when the organ is diseased or its functions perverted. As already described, the changes effected by the liver are separable into two main groups. On the one hand are those processes which deal with the various materials brought to the gland from the digestive tract, the proteid, carbohydrate, and

fatty products of digestion, as well as such substances as are distinctly toxic whencesoever derived, *i.e.* the nutritive, assimilative, and metabolic functions. On the other is the excretory or biliary function which is concerned with the manufacture and excretion of bile whereby certain products of proteid metabolism (bile acids) and of hæmoglobin destruction (bile pigments) are eliminated, subserving in the course of its removal definite accessory digestive functions. Effected as these several processes are by the hepatic cells and the epithelial cells of the ducts, alterations in these structures result in functional disturbance, whether restricted to certain of the processes or in a greater or less degree involving all; and that vague symptom-complex known as "biliousness" would seem to consist in a hepatic derangement in which both metabolic and excretory functions are in some measure upset. Within varying degrees, therefore, these several forms of hepatic activity may be independently or predominantly involved, and either the nutritive or the biliary functions may be alone affected.

The conditions to which these functional perversions are due are—(i.) Structural changes in the liver cells, especially those of a degenerative character, and that are induced by toxic agents, many of which are derived from the gastro-intestinal canal. (ii.) Catarrh of the biliary ducts. (iii) Congestion of the liver. (iv.) The febrile state. (v.) Excessive proteid food. There is no evidence of any direct interference with the hepatic functions from nervous causes, however probable such is the case; digestive disturbances so produced may indirectly affect the liver by leading to the formation of toxic substances.

Perversion of proteid assimilation.—That the liver effects some change in the absorbed proteids, and that in abnormal states of the organ these changes are often also abnormal, are equally certain. But much remains to be known as to the precise nature of the processes. There is no doubt that the greater part of the urea and some at least of the uric acid excreted from the body are formed in the liver, whether directly from the absorbed proteid digesta, or from those antecedents of these bodies (*e.g.* lactic acid and ammonia) which are derived from muscular and glandular activity. The bile acids are also evidence of the influence of the hepatic cells on proteid metabolism, since these bodies are derived from the decomposition of proteid substances, the hæmoglobin being probably the chief source. When the carbohydrates are excluded from the ingesta the proteid food may be in part split up in the liver into urea and glycogen or fat, or their precursors. Though not the only modes

in which the liver exercises an influence on the nitrogenous metabolism, they will serve to show how far reaching it is, and to suggest the many directions in which failure in hepatic function will give rise to symptoms referable to a perverted proteid assimilation. Whilst some of these symptoms are to be attributed to the circulation in the blood of toxic substances which have been formed in place of the more innocuous bodies normally produced, such as headache, lassitude, drowsiness, irritability of temper, muscæ volitantes, and palpitation, commonly comprised within the expression "biliousness," others are more closely connected with the digestion, such as nausea, heart-burn, loss of appetite, flatulence, and epigastric fulness, irregular bowels, dark or very pale stools; and others again are seen in the character of the urine, which may be small in amount and loaded with urates. Such a condition, when habitual, was denominated by the late Dr. Murchison "lithæmia," and was by him regarded as a manifestation of primary hepatic disturbance, and so to be closely allied to gout. But it is very doubtful how far such excess in the nitrogenous urinary solids is really expressive of primary impairment of the liver cells' function. For apart from the fact that much of the uric acid excreted is derived from certain articles of the food, and that leucocytosis, whether normal, as during digestion, or morbid, as in leukæmia, is also the source of much of this substance, there is reason to believe that the liver state which is associated with deposits of urates and uric acid is often induced as the result of faulty intestinal digestion, which gives rise to toxic substances, and these taken to the liver act as irritants to the organ in the course of their excretion into the biliary channels, at the same time determining a slight absorption of bile and consequent jaundice.

Perversion of carbohydrate metabolism.—This is shown by the presence of glycosuria or by the development of diabetes mellitus, conditions which have been described elsewhere (see Vol. II. p. 178).

Impairment of antitoxic power.—The ability to deal with the various poisonous bodies which reach the liver in the portal blood would appear to fail at times, and such substances consequently escape into the general circulation and produce their effects, many of which contribute to the state of "biliousness", or of so-called "torpid liver." The toxic materials may be of such a character as gravely to damage the liver cells, as is the case with phosphorus; and it is very possible that acute yellow atrophy of the liver, and some of the forms of infectious jaundice (Weil's disease) have a similar origin.

Variations in the biliary functions.—Very little is really known as to an excessive secretion of bile, exceeding, that is, the normal amount which varies under healthy conditions with the time of day, the amount of food, and especially of the fluid intake and other circumstances. The influence of drugs in promoting a secretion of bile has been much exaggerated, and few, if any, may be depended on for this purpose. For the popular notion that the symptoms collectively denominated "biliousness" are dependent on any such excessive secretion there is no warrant. No symptoms, indeed, can be attributed to any such excess, even if such excess occur; and any increase in the amount of bile in the stools is not to be taken as indicating a hypersecretion, but rather of an undue hurrying on of the upper intestinal contents.

A diminution in the biliary secretion is of more frequent occurrence and more easily recognised. The febrile state, certain poisons, such as phosphorus, arseniuretted hydrogen, snake poisons, and some bacterial toxins, lead to a decrease in the bile, and the same would sometimes appear to be the case in congestion of the liver. But although the total amount of bile that is found may be less than in health, the proportion of the contained pigments may be much higher than normal, inasmuch as most of the above-mentioned conditions cause an excessive destruction of red blood corpuscles and liberation of hæmoglobin, from which the bile pigments are derived. Although the red corpuscles are probably also the chief source of the bile acids, the proportion of these substances by no means necessarily corresponds to that of the pigments, and indeed they are often much diminished in amount when the colouring matters are in excess, indicating the independent activity of the hepatic cells in the formation of the bile constituents. Concerning the conditions which govern the formation of these acids much information is wanting.

Cholesterin, which forms so large a proportion of most biliary calculi, is now supposed to be formed and excreted by the epithelium of the bile ducts and gall-bladder rather than by the liver cells. It is kept in solution in the bile by the bile acids. Under conditions of catarrh of the mucous lining of the ducts and gall-bladder, such as would follow from any retardation of the bile flow owing to its increased viscidity or from elimination of toxic irritants by the duct-epithelium, a larger amount of cholesterin would be likely to be formed; and it is such conditions which are commonly associated with the formation of gall-stones, and that are best removed by flushing the liver with alkaline waters—as of Carlsbad, Harrogate, and

elsewhere—producing a more dilute bile and removing the excitant to the catarrh.

The urine in hepatic disorders.—Since the liver is so largely concerned in metabolic changes and in the processes concerned with the excretion of waste products, and the urine is the main channel of removal of such waste, it is only to be expected that disturbances of hepatic function will be manifested in the renal secretion.

Reference has been made to the diminished quantity of water and excessive uratic deposit met with in conditions of a large nitrogenous intake, especially if accompanied by much cutaneous secretion, or of an excessive nitrogenous metabolism with sweating, as in fever, and a similar result frequently supervenes on hepatic congestion such as follows obstructive heart disease.

The urine in these states is likely to be high coloured from an increased amount of pigment formed in the liver, from which are derived not only the normal urinary colouring matter, such as urobilin and urochrome, but also others which are probably abnormal, such as the pink and brick-red pigments (uroerythrin) so often seen in association with urates.

The normal bile pigments, biliverdin and bilirubin, are found in the urine as a necessary consequence of jaundice, which depends on the circulation of these substances in the blood after absorption from the bile passages. If there be jaundice there must be bile pigments in the urine, where they may be found even before the jaundice is apparent. But the presence of bile acids in jaundice is by no means so constant, and sometimes they are entirely absent, these two groups of substances being independently and not of necessity coincidently formed by the hepatic cells. Dependent on the presence or absence of bile acids in the urine a distinction of jaundice into hæmatogenous (*i.e.* of bile formed in the blood) and hepatogenous (*i.e.* bile formed in the liver) was formerly made—a distinction now known to be erroneous, since all jaundice is of the latter origin.

Pain in hepatic disease.—As with the solid viscera generally, extensive structural change may take place in the liver without giving rise to appreciable sensory symptoms, more especially if such changes have taken place gradually. A rapid enlargement of the organ, as from cardiac obstruction, will probably give rise to a dull sense of pain and heaviness and some tenderness on pressure. When the capsule and peritoneal covering become involved, then pain, sometimes very considerable, may be experienced in the hepatic area and

in the right loin. When the organ is very much enlarged a feeling of weight is perceived, and the patient cannot lie on the left side without discomfort owing to the falling away of the viscus and the strain on the suspensory ligaments. A large abscess or cyst, or an extensive lardaceous degeneration, or an advanced cirrhosis with shrinking of the organ, may occur without any disturbance of sensation, and even without giving rise to tenderness on deep pressure. As a diagnostic sign, therefore, of hepatic disease pain is of very slight significance. Not infrequently, however, a suggestive symptom is to be found in pain referred to the right shoulder (fourth cervical area), which is frequently to be met with in disorders of the liver, more particularly those of an acute character, such as congestion and abscess.

The intense pain connected with the passage of a biliary calculus will be referred to later.

Pyrexia in hepatic disease.—There is scarcely any affection of the liver that may not be accompanied by a rise of temperature, though the character of its course is marked by no special constancy in duration or degree. Even in hepatic abscess the extent of the fever is most variable (p. 441). In the so-called hypertrophic cirrhosis an evening rise to 103° or thereabouts is usual, whilst in the atrophic form the pyrexia is much less frequent, and may only be noticed after long-continued observation. Of somewhat more importance is the occurrence of attacks of high temperature with rigors in cancer of the liver, closely resembling a similar condition which develops with greater frequency in cases of impacted gall-stone. Hence but little help is to be obtained from a consideration of this symptom in making a differential diagnosis between these conditions, agreeing as they do in the presence of jaundice and a distended gall-bladder. The pyrexia is doubtless of septic causation, aided possibly in the calculous affection by reflex nervous irritation.

JAUNDICE

By jaundice or icterus is meant a staining of the integuments, mucous membranes, and tissues generally by the bile pigments, due to the circulation of these substances in the blood, consequent on their absorption from the biliary passages. The cause of the absorption is in all cases some obstruction in the channels which interferes with the proper escape of the bile into the duodenum, whether this be in the main ducts or in the finest bile capillaries.

Pathology.—It is to be distinctly remembered that the proper bile pigments are only formed by the hepatic cells, and are not simply excreted by them from the blood, nor is there any evidence that the hæmoglobin when liberated from the corpuscles in the blood (hæmoglobinæmia) can be decomposed into bile pigments in that fluid. All jaundice, therefore, is hepatic in origin (hepatogenous), and a hæmatogenous jaundice has no existence, though certain blood states—toxæmias—may favour the occurrence of jaundice partly by causing an increased hæmolysis, and hence a greater supply of the material from which the pigments are formed, and partly by setting up a catarrh in the intrahepatic ducts by the irritant nature of the poisonous substances excreted by the liver cells. This catarrh determines an increased viscidity of the bile, owing to less water being secreted and more mucus, and hence some obstruction to the bile flow is produced. Directly any obstruction exists, a transudation of the bile takes place from the ducts into the lymphatic spaces and vessels, the pressure in which is exceedingly low, and so reaches the blood current. When the obstruction involves the finest bile capillaries the bile would seem to pass directly from the hepatic cells into the lymphatics, and when that is the case the larger channels and gall-bladder may contain an almost colourless mucus, which was erroneously regarded as evidence that the jaundice was due, not to the liver, but to changes in the blood. That the liver cells still retained their functional capability in these circumstances was as unknown as that the finest channels were obstructed, and such cases were known as "jaundice from suppression"—the liver being supposed to have ceased its activity in respect to the biliary function. Nor is there any good ground for the belief formerly entertained that jaundice was sometimes due to an excessive out-pouring of bile into the intestine and its subsequent absorption therefrom—"jaundice from polycholia." In these cases of jaundice the fæces retained their normal colour—a condition which is capable of quite a different explanation, such as only a partial obstruction of the channels sufficient to raise the bile pressure enough to effect its transudation into the lymphatics, but not complete enough to exclude all bile from the bowels. There is no proof whatever of a polycholia ever occurring, though a bile unduly rich in pigment (polychromia) is a very frequent result of toxæmia for the reason already given.

Notwithstanding, however, that jaundice is always hepatogenous and obstructive in origin, it is clinically convenient to distinguish between those cases where the obstruction is obvious and affects the

largest ducts, and those—sometimes called "hæmohepatogenous"—where the primary underlying cause is a toxæmia, the obstruction and consequent retention and absorption of the bile from the smallest passages being secondarily induced. To the former group the term "obstructive" is frequently though improperly restricted, and to the latter the definition "toxæmic" is applied; and with this understanding of their significance these terms will for convenience be employed here.

The conditions leading to obstruction of the main ducts (hepatic and common bile duct) may be thus grouped:—

i. Foreign bodies within the ducts.

Gall-stones or inspissated bile.

Ascarides or other parasites that have obtained entrance from the bowel.

ii. Changes in the walls of the ducts.

Catarrhal swelling of the mucous lining, often extending from the duodenum, though sometimes primary. (When affecting the finest channels and induced by noxious irritants, this condition is the essential cause of jaundice in toxæmia.)

New growths of the duct, or of the ampulla of Vater.

Stricture of duct from cicatrix. Closure of orifice from healed duodenal ulcer. Congenital obliteration of the duct. Spasm of the duct has been suggested as a cause of so-called emotional jaundice, a very rare occurrence that has been known to occur in association with great anxiety, terror, and the like.

iii. Pressure on the duct from the outside by tumours connected with adjacent structures, aneurysm, and even by fæcal accumulation in the transverse colon, or by pregnancy or other uterine enlargements.

It is possible that the obliquity of the course of the terminal portion of the common bile duct through the wall of the duodenum facilitates its obstruction by any of the above-mentioned conditions.

The toxic states which may be accompanied by jaundice are poisoning by—

i. Phosphorus, arsenic, antimony, mercury, toluylenediamin, chloroform, and ether.

ii. Snake venom.

iii. The toxins of most of the acute infective diseases, more especially yellow fever, relapsing fever, malaria, typhus, Weil's disease, icterus gravis, and acute yellow atrophy of the liver, and pyæmia.

Although all these poisons cause, among other results, destruction of the red corpuscles, their power in producing jaundice is not proportional to their effect in this direction, and there are some morbid states associated with extreme hæmolysis, such as pernicious anæmia, in which jaundice is entirely wanting. Clearly, therefore, the symptom is that of individual specific poisons, rather than of all those of a hæmolytic character.

The consequences of bile circulating in the blood vary somewhat with the cause of the absorption, whether obstructive or toxæmic. Certain features, however, are common to both.

a. Staining of the skin and mucous membranes.—The extent to which this—the essential feature of jaundice—takes place differs with the completeness of the obstruction, its cause, its duration, and in some measure in the complexion and amount of subcutaneous fat of the individual, as well as the existence or otherwise of any cyanosis. The coloration varies in these several conditions, from a faint lemon-yellow tinge—first noticed in the conjunctivæ, and in the mildest cases limited to that situation—to a deep orange, or a dark olive green in long-standing obstruction. As a rule the pigmentation is much less in toxæmic states, the deepest staining being met with in permanent closure of the common duct by malignant disease or by gall-stone. Some fluctuation in the colour is frequently noticeable from time to time, presumably in accordance with the activities of the liver and of the kidneys, by which the pigment is eliminated. Speaking generally, the jaundice is deeper in those of sallow complexion, in the thin rather than in the stout, and in the old rather than in the young.

Care must be taken to discriminate true jaundice from pigmentation due to other causes, such as the bronzing of the skin from Addison's disease, or from exposure to the sun. Patches of subconjunctival fat may be mistaken for icteric staining. Malaria, anæmias, especially the pernicious form, and the malignant cachexia, each tends to produce a tinting of the skin of various shades of yellow or brown resembling jaundice.

It is also to be remembered that the coloration of the skin may remain for some time after the cause has been removed, gradually disappearing as the pigment is eliminated; and also that unless very deep it is not recognisable in artificial light.

Post-mortem every vascular tissue is found more or less bile-

stained, even the bones, the liver being deeply mottled when the cause is obstruction of a main duct.

b. Coloration of the secretions.—If there be bile pigment in the blood it will also be found in the urine; hence this fluid is always coloured in jaundice very much in proportion to the degree of cutaneous pigmentation, and varies from a slightly darker tint than normal to a deep greenish black colour. It may be perceptible with the nitric acid test even before the conjunctivæ are discoloured, and when the causal obstruction is very temporary the pigments may be recognised only in the urine.

Although the pigments are invariably found in the urine in jaundice, the same is not the case with the bile acids, which are deficient in that of toxæmic causation, being in largest quantity when the obstruction involves the main ducts.

Renal casts stained yellow have been seen in the urine.

The sweat, the milk, and still more rarely the saliva and tears, have been known to contain traces of the pigment, in the cutaneous secretion enough even to discolour the body-linen.

c. Various *skin eruptions* have been noticed in association with jaundice, such as urticaria, lichen, and boils, but they are far from constant. In long-standing cases of moderate intensity yellowish plaques or nodules appear in the skin, especially in the eyelids, though also occurring in other parts of the face, and on the extensor surfaces of the limbs—known as xanthelasma or vitiligoidea. Occurring oftenest in middle-aged women, this affection is most commonly associated with jaundice or other hepatic disorders, though not invariably so, and its pathogeny is but imperfectly understood. Subcutaneous petechiæ are also very common in chronic jaundice.

d. A not infrequent accompaniment of obstructive jaundice, though not observed in the toxæmic variety, is *pruritus* apart from any eruption. It is very irregular in occurrence, and may precede the actual jaundice. The itching is worse at night.

e. Yellow vision.—Very occasionally white objects appear yellow to jaundiced patients, a phenomenon that appears to be due to some disturbance of the nervous visual structures, and not to staining of the tissues of the eyeball, as formerly supposed. This symptom is also only met with in obstructive jaundice.

f. A bitter taste is sometimes experienced, possibly due to the presence of taurocholic acid in the saliva, but it is not restricted to those affected with jaundice.

g. In jaundice, unaccompanied by fever, it is usual to find the *pulse* much diminished in frequency—even to 30 per minute, or

lower—and of lessened tension. This is said to be due to the inhibitory effect of the bile acids on the heart. The slowness is not observed in febrile cases, which are usually toxæmic, and in which the bile acids are more or less absent, nor is the symptom so marked in children as in adults.

h. Under certain circumstances, not altogether understood, patients suffering from jaundice, more especially the toxæmic form, may pass into a grave and fatal condition, apparently due to some poison connected with the bile retention, or to the arrest of the eliminative function of the liver, whereby toxic substances formed in the intestines and elsewhere, and which should be excreted by the hepatic cells, are not thrown off. This state is sometimes termed *cholæmia*, a description of which has been given in Vol. II. p. 87. The onset of this state is commonly marked by nervous symptoms, drowsiness, delirium, or coma. Jaundice, however, may persist for many months, or even years, without any such manifestations.

The results of the exclusion of bile from the bowel are chiefly apparent in the character of the stools, which are colourless and clay-like in proportion to the completeness of the exclusion. Inasmuch, however, as the secretions from the bowel are likely to be bile-stained, care must be taken not to mistake any superficial coloration such fluids may cause with the proper pigmentation of the fæces.

Since the bile is an adjuvant to the pancreatic secretion as a digestive agent, and is a very mild antiseptic, and also a very moderate excitant of peristalsis, its absence from the bowel is apt to cause various disturbances of intestinal digestion. But the degree of these varies much. Constipation often coexists with jaundice, but not always, the stools may be ill-smelling or almost odourless, and the presence in the motions of undigested fat is seldom seen as the result of bile absence only. Other coincident conditions are probably more responsible for these symptoms, the last named being far more commonly due to an exclusion of the pancreatic secretion from the bowel, which will occur if the causal mischief involve the common orifice of the ducts in the duodenum. Any considerable emaciation in association with chronic jaundice rather betokens malignant disease, or absence of pancreatic juice, than lack of bile only.

It is of great importance accurately to ascertain the colour of the stools in every case of jaundice as a measure of the completeness of the obstruction. Where the primary cause is toxæmia the motions are pigmented, since the channels are but partially blocked, and there is an excessive formation of pigment from the increased

hæmolysis. Or the stools may be intermittently coloured, such as occurs when a calculus is loosely lodged in the ampulla of Vater, acting as a ball and socket valve.

Icterus neonatorum.—A large proportion of infants manifest a transient slight jaundice coming on a day or two after birth, and completely passing off within a week—rarely lasting longer. In contrast to what obtains in later life, the sclerotics are much less tinged than the skin and may entirely escape, and are seldom affected before the integuments. The fæces exhibit the normal coloration.

No altogether satisfactory explanation of this phenomenon has been given; but it seems probable that it is in some way associated with the sudden and violent disturbance of the circulation, particularly through the liver, which takes place at birth, and it is affirmed by Quincke as being dependent on a prolonged patency of the ductus venosus allowing some of the portal blood into which bile has been absorbed from the bowel to pass into the general circulation.

Except when due to a congenital occlusion of the common duct, or the cirrhosis of hereditary syphilis, the jaundice of infants soon clears up and requires no treatment.

The **treatment** of jaundice, which is but a symptom of many morbid conditions, should be directed as far as possible to the cause, to the ascertainment of which every effort should be made. For the actual removal of the coloration of the integuments very little can be done, but it is desirable to assist so far as possible the elimination of the bile constituents by the kidneys, and for this purpose the drinking of indifferent or alkaline mineral waters, or even plain distilled water, should be encouraged to wash out the renal tubules. To the same end, and also as assisting in relieving any portal engorgement or intestinal catarrh, the bowels should be kept open by a daily saline aperient such as Carlsbad salts, or sulphate or phosphate of soda, with the occasional addition of a dose of calomel or blue pill made up with colocynth or rhubarb. A daily large simple enema is often very beneficial. Drugs reputed to possess a cholagogue action, such as euonymin, podophyllin, salicylates, and benzoates, are sometimes useful when the obstruction in the ducts is but moderate and of catarrhal origin. The digestive disturbances so often associated with jaundice may be in some measure corrected by an alkaline or acid and bitter mixture (*e.g.* ℞ Sodii bicarb. gr. xx. : Sp. chloroformi ℥x. ; Infus. gentianæ vel calumbæ vel chirettæ ad ℥j. M. ft. haust. To be taken between meals). Or

in place of the alkali, acidi nitro-muriat. dil. ♏x. may be given with the infusion, with some advantage if the tongue be not coated. Baths containing nitro-muriatic acid sometimes give relief, especially from the itching, for which purpose also sponging the skin with vinegar or lemon juice and water, or with a lotion containing hydrocyanic acid ʒj., borax ʒj., and cherry laurel water to ℥viij. Subcutaneous injections of nitrate of pilocarpine to induce profuse sweating may be tried. This symptom may be so troublesome as to necessitate the giving of an opiate. The use of ox bile in 5 to 10 gr. doses in capsules to make up for the absence of bile in the bowel is of doubtful value. The diet generally should be light, and it is well to avoid much fat except in the form of milk; alcohol also should be withheld.

W. H. Allchin.

SPECIAL SYMPTOMATOLOGY AND TREATMENT OF HEPATIC DISEASES

CONGESTION OF THE LIVER

By this is meant an abnormal hyperæmia of the liver, which may be transient or persistent, and, if persistent, may lead to important structural changes in the liver substance.

There are two distinct kinds of congestion of the liver, active and passive. It is called active congestion when the excess of blood is derived from the portal vein and hepatic artery, and passive when it is due to obstruction to the flow of blood from the hepatic veins. These two varieties are widely different in their causes, symptoms, and results, and must be considered separately.

ACTIVE CONGESTION

Hyperæmia of the liver occurs normally during the active period of digestion, during and after each meal, in a way common to all glands whose function is in any degree intermittent, and the variation in the amount of blood which the liver is capable of containing is very considerable. During this active period of the digestive process the liver undergoes distinct increase in size; but this normal hyperæmia, called forth by the periodic exercise of its functions as

one of the organs of digestion, is not congestion. Only if there be any excess of hyperæmia at that time, or if it persist after the active period of digestion is over, does it become congestion in the clinical sense, and it is then associated with more or less clear evidence of disordered function and malaise.

In the normal hyperæmia of the liver during digestion, and in all forms of active congestion from whatever cause, we must presume that the portal vein takes the principal, and the hepatic artery only a subordinate part, the amount of blood which the latter is capable of delivering being comparatively small; and it will be found that the chief causes of active congestion, and its consequences, are conditions affecting the viscera from which the portal vein takes its origin.

Etiology.—The causes of congestion of the liver are dietetic, gastro-intestinal, climatic, and toxic.

The *dietetic* causes are those which make an exaggerated call on the normal functions of the liver by excess, and especially habitual excess, in food and drink, and more especially in rich, highly-seasoned dishes in unnecessary quantity and variety, as well as in alcoholic drinks. Excess in quantity is just as likely to induce congestion as error in quality as regards food, and experience points to ardent spirits and the stronger malt liquors as more potent causes of congestion than the lighter wines, more especially if taken on an empty stomach. The habit of drinking large quantities of fluids at meals, so prevalent in hot climates, though they dilute the alcohol for which they form the vehicle, is also to be deprecated.

Such errors in diet on the side of excess are doubly effective if at the same time little or no exercise be taken.

The causes connected with the state of the *gastro-intestinal* canal are constipation and catarrhal and ulcerative conditions of the mucous membrane of the bowel. The congestion of the liver, which is sometimes described as a concomitant of dysentery, is, however, more likely to be a local hepatitis induced by septic absorption.

Climatic causes.—A study of the geographical distribution of hepatitis shows that it is far more frequent in hot than in temperate and cold climates, and the same can be said of all active hyperæmias of the liver. The causes of this greater frequency are not far to seek. Under tropical conditions a relatively large quantity of blood is habitually directed to the surface of the body, which prevents a rise of body temperature above normal limits under atmospheric conditions which render this a matter of considerable difficulty, as when the air is loaded with moisture, and its temperature

approaches or exceeds that of the body. A large volume of blood circulating at the surface is then required for purposes of efficient radiation, and to supply the fluid necessary for the profuse perspiration which is poured out to facilitate evaporation. The habitual presence of this large quantity of blood at the surface, and the free perspiration, render the resident in hot climates exceedingly susceptible to chills, and as the seasons in which the provision for surface cooling is most active are also those in which sudden changes of atmospheric temperature are most frequent, and the smallest thickness of clothing worn, a revulsion of blood from the surface to the internal organs is of frequent occurrence. The blood thus sent in from the surface finds a place in the portal veins and liver. Transient attacks of congestion of the liver under such circumstances are of frequent occurrence, and may even occur in temperate climates.

When the effect of repeated chills is added to those of excessive indulgence in rich food and strong alcoholic drinks, the commonness of hepatic congestion in hot climates requires little further explanation.

Toxic causes.—But the frequency of congestion of the liver in the tropics is not entirely due to these causes. A most potent and frequent cause is the prevalence of malarial fever. Every recurrence of fever of this kind is accompanied by congestion of the liver, and as these recurrences may take place every day, or every second or third day, for several weeks, a condition of more or less permanent congestion with enlargement of the liver results. In such cases the spleen is also invariably enlarged. The same condition of the liver takes place, in fact, in all febrile conditions in all climates, and even in animals whose body temperature is artificially raised, it seeming to be an effect of mere elevation of body temperature, apart from any result of specific infection; but in malarial fevers there is a special tendency to congestion of the liver and spleen, as evidenced by the deposits of melanin found in these organs after such attacks, showing that these organs have a special relationship to the parasite. The other febrile conditions which especially cause congestion of the liver are febrile jaundice, icterus gravis, yellow fever, Weil's disease, and black-water fever; but it may be said to be a part of all continued elevations of body temperature.

Age and sex.—Congestion of the liver is most frequent between the ages of twenty and thirty-five, and is more common in men than in women, on account of the grosser habits of life of the former, and their greater exposure to exciting causes.

Symptoms.—The symptoms of an acute attack of congestion of the liver are uneasiness or pain in the region of the liver, and

perhaps at the back of the right shoulder, dull headache, anorexia, a foul tongue, constipation, a muddy complexion, and some yellowing of the conjunctiva. In toxic cases the jaundice may be intense. At the same time the urine is scanty, of high specific gravity and colour, and it may deposit urates copiously.

On examining the liver it will be found to be uniformly enlarged, and its lower margin is felt an inch or more below the costal arch in the right nipple line, the measurement of absolute dulness being increased to five or six inches. Palpation in this position will give rise to pain, as will also percussion over the exposed portion of the liver ("the congestive spot").

In chronic hepatic congestion the general malaise is not so great, the tongue may be clean, or nearly so, and there may be little or no pain in the region of the liver. There is always constipation, and piles are generally present. The appetite is capricious, the temper irritable, and there is mental despondency.

The liver will be found to be uniformly enlarged, and tender to percussion and palpation, with a firmer consistency than normal.

In cases of malarial origin there are, even when the patient has come to live in a temperate climate, frequent rises of temperature, which may be of less than twenty-four hours' duration, or may constitute a febricula lasting two or three days, or they may be distinctly intermittent for a similar period. Each such rise of temperature is accompanied by a recrudescence of the liver symptoms.

Prognosis.—In non-toxic cases, and even in malarial cases, the prognosis is favourable in recent attacks, provided the causes be removable and appropriate treatment adopted. In chronic cases the condition is seldom one of pure congestion. Trophic changes in the hepatic structure occur early in these patients. If these are confined to hypertrophy of the glandular structure complete recovery may still be expected, but if they include proliferation of the connective tissue perfect restoration of the liver to its normal condition and functions cannot be looked for. The effect of treatment in reducing the size of the liver will be the inverse measure of the degenerative changes which may have occurred.

Treatment.—All cases must be placed on a simple non-stimulating diet, and alcohol in any shape absolutely forbidden. A mercurial purge, followed by a saline draught, gives the most rapid relief to the engorged portal system. Poultices and mustard plasters may be liberally applied after the bowels have been opened. The patient, especially if he have lived in the tropics, should on no account be allowed to bathe in cold water. He should be kept in

bed in a warm room till the acute symptoms have passed, and the saline draught should be repeated twice or three times a week for some time.

In the more chronic cases the same rules as to diet must be enforced, with a dose of Carlsbad salts every second or third morning, together with an acid tonic mixture three times a day, and in cases of malarial origin this may be combined with arsenious acid. Patients subject to recurrent attacks of acute congestion will derive much benefit from a visit to Carlsbad or Homburg, where they will be able to carry out the necessary *régime* with less difficulty and smaller temptation to commit irregularities in diet. They must take a fair amount of gentle exercise, clothe warmly, and avoid chills. A teaspoonful, more or less, of Carlsbad salts should be taken in the early morning in hot water, with the object of attaining two loose motions, one before and the other after breakfast. The poultices must be of large size, 12 inches broad, 2 inches thick, and extending from the left nipple line round the front of the chest to the spine behind. If the congestion be dependent on active malarial poisoning, indicated by rises of temperature and the presence of parasites in the blood, quinine should be given regularly and methodically till these symptoms have passed away.

In chronic cases which have resulted in hypertrophic or other structural changes in the liver, a number of successive blisters will be found useful.

Chloride of ammonium has been much praised in the treatment of the acute stages of hepatic congestion, in twenty-grain doses three times a day; but its usefulness is doubtful, and it should not be substituted for the treatment here laid down.

PASSIVE CONGESTION OF THE LIVER

Passive congestion of the liver results from resistance to the flow of blood from the hepatic veins into the vena cava inferior. It may be caused by bending of the latter vessel as the result of pressure and displacement, owing to the presence of a large quantity of fluid in the right pleural cavity, or it may be due to obstructed circulation in the lungs from chronic pulmonary diseases; but the most frequent cause is valvular disease of the heart itself, especially stenosis of the mitral orifice, with distension of the left auricle, impeded pulmonary circulation, and consequent stress in the right ventricle; but mitral regurgitation, and indeed any condition which

ultimately leads to difficulty in the right side of the heart, will cause passive congestion of the liver.

Symptoms.—The hepatic dulness on percussion will be found increased in all directions, and in heart cases the enlargement is often very great. It takes place more rapidly than in active congestion, and in the early stages there is sometimes expansile pulsation, both palpable and visible, synchronous with the heart's action. Later, when induration has taken place, this is no longer possible, but pulsation may still be communicated to the liver by the abdominal aorta.

The obstructed flow in the central veins of the lobules prevents free circulation in the portal system, and gastric and intestinal catarrh, diarrhœa, or constipation and piles, are the result. Jaundice is frequent, and sometimes intense, and a peculiar greenish hue is imparted to the countenance from the mixture of the yellow of the jaundice with the blue of the cyanosis of the original cardiac or pulmonary disease.

Ascites appears early in passive congestion of the liver, but it does not, as in active congestion, necessarily mark the supervention of cirrhotic contraction, as it may be a part of the general dropsy caused by the disease on which passive congestion depends.

The prognosis is that of the disease which is the cause of the condition of the liver.

Treatment.—For obvious reasons treatment must be directed to the original disease. The portal system may be relieved by wet cupping or leeches over the hepatic area, by occasional saline purges, and the diet should be of a kind suitable in gastro-intestinal catarrh.

HEPATITIS

Definition.—Inflammation of the parenchyma of the liver, frequently ending in suppuration.

The term is often loosely used in reference to cases of enlargement and engorgement of the liver, the result of repeated attacks of malarial fever, with or without alcoholic complications; or to the early stages of hypertrophic cirrhosis, with accompaniments of pain, and perhaps febrile symptoms; or to the results of congestion of whatever cause. It is also assumed by some systematic writers to be the basis of all parenchymatons changes in the liver, and they consequently divide it into—(*a*) *epithelial hepatitis*, where the changes take place primarily and chiefly in the secreting cells, as in

phosphorus and other poisoning, in the eruptive or other infectious fevers, yellow fever, relapsing typhoid, black-water fever, icterus gravis, acute yellow atrophy, etc.; and (*b*) *fibrotic hepatitis*, in which the changes primarily affect the connective tissue framework of the organ, as in alcoholic, paludic, and syphilitic cirrhosis, and in suppurative hepatitis of venous or arterial origin. Here it is confined to true inflammation of the substance of the liver, generally caused by infection by septic particles reaching the liver from an inflamed or ulcerated portion of the bowel or elsewhere, which may resolve without the formation of pus, or pass on to suppuration.

The diagnosis of non-suppurative hepatitis is extremely difficult, and it often occurs and advances to abscess without betraying any recognisable indications of its presence. It would be distinguished from malarial or other engorgement by there being less general enlargement of the liver, by the pain being more acute and localised, especially if the inflamed focus were situated near the peritoneal covering, which would then participate in the inflammation, and by palpation eliciting pain at a part of the liver other than that palpated. The rectus muscles would also be more "on guard" in inflammation than in congestion, especially if the focus of inflammation were in the left lobe. From suppurative inflammation it can only be distinguished by complete subsidence of all symptoms under treatment.

Treatment.—The treatment does not differ materially from that of acute congestion. The patient should be put to rest in bed, with hot poultices or fomentations of large size over the liver. At the same time everything should be done to lessen active congestion by a very light diet, abstinence from alcohol, and brisk saline purgation.

HEPATIC ABSCESS

Definition.—Suppuration of the parenchyma of the liver, of most frequent occurrence in hot countries, and very often associated with dysenteric ulceration of the intestinal mucous membrane.

Pathology and morbid anatomy.—It is often possible to study the whole process of suppuration in a single section of liver tissue. It begins by the formation of groups of congested spots of a port-wine colour. In those which have not yet begun to break down the first change is seen to take place in the trabecular supports of the glandular tissue. They become enlarged, rich in nuclei, and infiltrated with oily drops. Each suppurating focus is next seen to consist of two parts—a central, anæmic, undergoing necrosis and

softening, and marked by an accumulation of leucocytes in the capillary network of the lobules; and a peripheral, hyperæmic, and consisting of the surrounding tissues gorged with blood in dilated vessels. In the centre appears a pulp of softened gland structure, consisting of amorphous matter, derived apparently from the protoplasm of the hepatic cells, some of which may still be recognisable tumified, provided with several nuclei, and occupied by fatty globules, leucocytes, and various microbes.

The macroscopic appearances correspond with those just described. The centres of several small spots of hyperæmia of the hepatic parenchyma, grouped round one point, become yellowish or whitish, with a surrounding purple areola of hyperæmia. These spots at first, perhaps half an inch in diameter, and separated by dark discolorations, or incompletely united by fibrous connections, gradually increase in size by union with adjacent foci. Their softened centres contain a dirty sero-purulent or frankly purulent fluid, more or less deeply tinged with red from admixture of blood. By continued softening and breaking down of intervening tissue these purulent spaces burst into one another, till, from being the size of a small nut, they become as large as an orange or a cocoa-nut, of very irregular shape, with fingers of still consistent liver tissue projecting into their interior. In acute abscess of the liver this process of destruction is a very rapid one, and the abscess may reach a large size in a few days; but the process is often slow, and in that case the abscess becomes more regular and globular in shape from destruction of the finger-like protrusions into the interior; it may even come to possess a smooth pyogenic lining membrane, and in that phase it may retrograde and dry up, leaving only cicatricial evidence of its former existence.

The bacteriology of hepatic abscess is in an unsatisfactory condition. Its very frequent association with, and obvious dependence on ulceration of the bowel, or on some septic condition existing elsewhere, point strongly to the conclusion that it originates in every case in the importation of septic material or organisms by the portal or hepatic capillaries; organisms of this nature are habitually found in the walls of the abscess and their hyperæmic surrounding zone, and they have even been detected in thrombi, which have formed in the small divisions of the hepatic vein in the neighbourhood of a suppurating focus. The organism most frequently found is the amœba coli, which is present in about one-half of the cases; and in addition to those organisms more usually associated with suppuration elsewhere, namely, streptococci and staphylococci, the

bacillus coli communis may also be found in the ragged walls of the cavity. The frequent association of the amœba coli with dysenteric ulceration of the bowel, and with hepatic abscess, is very remarkable; but it does not necessarily follow that it is the cause of the condition in either case, as it is a normal inhabitant of the intestine, and it may only find in these diseases conditions favourable to its growth and multiplication; or in hepatic abscesses it may serve the part of a transport animal for the really active septic material which determines the suppuration. The pathology of dysentery is at present too chaotic to enable us to say whether "amœbic abscess of the liver" is more frequently associated with "amœbic dysentery" than with the bacillar variety of that affection, or whether the "bacillus dysenteriæ" will yet be found in those cases of liver abscess of dysenteric origin in which the amœba coli is not present. Such questions can only be answered by a more exhaustive examination of the fauna and flora of these diseases than has yet been satisfactorily carried out.

But however that may be, and while the walls of a hepatic abscess may be alive with amœbæ and other organisms, its purulent contents are usually sterile. It is quite exceptional to find them otherwise, when they are withdrawn by an aspirator, or when the abscess is first opened. It is only after a few days, when the abscess walls are breaking down, that living amœbæ and other organisms appear in the discharges. The cause of this sterility is doubtless something unfavourable to their existence in the character of hepatic pus, which makes it impossible for them to live except in the abscess walls, where the process is more or less slowly advancing, and where they are still protected by living tissues from the contained pus. That it is not due to mere chronicity is proved by the fact that the pus of acute abscesses of the liver, of a few days' or weeks' duration, is equally sterile, if we may judge by the impunity which follows the escape of such pus into the peritoneal cavity during an operation.

The presence of an abscess generally, though not invariably, causes an increase in the dimensions of the liver. In one hundred and one autopsies Rouis found it normal in size in twenty-eight, augmented in seventy, and diminished in three. In those of normal and diminished volume the purulent contents of the abscess did not exceed 250 grammes, but in the others the quantity was greater, the tumefaction being in proportion to the dimensions of the abscess and the degree of general congestion. The abscess cavity may practically occupy the whole of the liver substance with small ex-

ception, and it may fill a space from the fifth rib to the crest of the ileum. The largest abscesses are naturally found in the right lobe.

Hepatic abscess being usually the result of the fortuitous dissemination of septic matter by the blood of the portal vein, any point of the liver may be the seat of suppuration, and it occurs in the different parts of the organ in frequency proportionate to their size. Thus Rouis found the right lobe affected one hundred and fifty-four times, the left lobe thirty-three times, and the Spiegelian lobe nine times. The number of foci of suppuration probably depends on the severity and chronicity of the intestinal disease, which is the most frequent source of the infecting material. In the majority of cases the abscess is single, but among fatal cases there are a large proportion of multiple abscesses. Rouis found single abscesses in 75 per cent, multiple in 25 per cent, of post-mortems; Dutroulau 68 per cent of single, and 32 per cent of multiple, abscesses. In the returns of the Government of India for 1899 the figures are 65 and 35 respectively. In multiple abscess the different abscesses are sometimes synchronous, but oftener they are of different dates, and some may generally be seen in which the process which ends in abscess is only beginning, suggesting more recent infection, while in others suppuration has been in existence for some time. In multiple abscess the number may be from two to six, with a tendency to run into one another, when the multiplicity diminishes at the cost of more and more liver tissue.

Etiology.—The influence of climate and season cannot be questioned. The maximum frequency of this disease corresponds to a zone comprised within the isotherms of 75° or 80° Fahrenheit. Wherever these mean temperatures are reached and exceeded, hepatic abscess appears as a cause of mortality in increasing frequency; and in regions coming under such climatic conditions the frequency and mortality from this cause are greater in the hot than in the colder seasons. In Algiers, for instance, the number in the hot season has been as much as 185, while in the cold season it fell to 89. It must be remembered, however, that heat has probably only an accessory influence, and that the essential cause of this greater seasonal prevalence is the relative prevalence of dysentery at the same seasons, for all hot countries are not equally noted for the occurrence of hepatic abscess. Pondicherry, for example, thanks probably to its magnificent water-supply, is remarkable, as compared with the surrounding districts of Madras, for its immunity both from dysentery and hepatic abscess. It is also less frequent in the West than in the East Indies, but, as a rule, it prevails within the isotherms

noted above, whether these remain within the tropics or extend beyond them. It is more prevalent in Madras than in Bombay and Bengal; more frequent in Shanghai, Canton, and Hong Kong than in Japan. It is met with in Northern Australia within the tropics, but is rare in the more southern parts; common enough in Northern Africa and Egypt, and on the east and west coasts of tropical Africa; it is seldom seen in the Cape Colony, which is beyond the 70° isothermal line, following generally in all these countries the distribution of dysentery. It is rare in Northern Europe, but less so in Italy and the more southern parts.

Race.—All races are not equally liable to hepatic abscess. A comparison of the returns for the army of India shows that the mortality from abscess of the liver is thirty-two times greater among European than among native troops, and this notwithstanding the greater prevalence of dysentery among native soldiers. Among native regiments hepatic abscess occurs chiefly among Ghurkhas and Madrasis. In 1899, of the eight admissions among native troops, five occurred in Madras and two in Ghurkha regiments. It is not improbable that liver abscess, like tuberculosis and enteric fever, is more frequent in the native army than these figures would lead one to suppose, because of the difficulty of diagnosis in many cases, and the absence of autopsies. It is doubtful whether the negro races enjoy a similar immunity.

Length of residence.—Though the older resident is not exempt, hepatic abscess is most common among recent arrivals, and especially those under six years' residence.

Age and sex.—Although equally subject to dysentery, children hardly ever, and women very rarely suffer from hepatic abscess. A medical officer may spend his life in the tropics without seeing a single case in a woman or a child, though he may never be without a male patient suffering in that way. It is most frequent in males between twenty and thirty-five years of age.

Alcohol.—The exemption of women and children, and the infrequency of hepatic abscess among natives of India who abstain from alcoholic stimulants, led at one time to the opinion that alcohol was the chief cause of the disease, and it must be admitted that all the evidence is in favour of the belief that it is a potent predisposing cause, along with others capable of inducing hyperæmia (congestion) of the liver. Waring found that 65 per cent of his cases of liver abscess occurred in alcoholics. A liver whose tissues have lost their resisting power by chronic congestion, or repeated attacks of acute congestion, whether these are the result of acute or chronic

alcoholism, or of other dietetic excesses, is more likely to fail at the critical moment to throw off some septic particle reaching it from the intestines or elsewhere, than a liver whose resistance to infection has not been impaired in that way.

Chills.—In a similar way chills are never the cause of abscess of the liver, though they may weaken, by the internal congestion they cause, the resisting power of the liver against septic infection, for the time being.

Dysentery and other septic foci.—Ever since the time of Twining and Annesley the association of hepatic abscess with dysentery has been recognised, but the discovery of the amœba coli, and its presence in the pus and walls of liver abscesses, as well as in the evacuations and ulcerations of many cases of dysentery, has raised the connection between the two conditions from one of mere association to one of probable cause and effect. But dysentery is not the sole cause of liver abscess; any septic suppuration of the bowel or pelvic organs may give rise to suppuration in the liver through the medium of the portal system, and, through that of the hepatic artery, it may be the site of pyæmic abscess, having a similar origin in any other part of the body. Thus it may result from any ulceration of the digestive canal, wherever situated, as ulcer of the stomach or duodenum, appendicitis, typhoidal ulceration of the ileum, abscess of the rectal wall or prostate, or peri-uterine suppuration following parturition, aseptic ulceration of the umbilicus in the newly born, or it may add a still graver import to ulcerative endocarditis; but these causes of suppurative hepatitis are altogether subordinate in frequency to dysentery, which, from the number, depth, and nature of the ulcers in that disease, and the large extent of intestinal surface involved, offers the most favourable conditions for the formation of septic material, and for its absorption by the portal radicles.

A very large proportion of patients suffering from tropical abscess of the liver are able, on careful inquiry, to give a history of previous dysentery. Neil Macleod found in forty cases positive evidence of previous dysentery in thirty-nine, and if the inquiry be not limited to recent dysentery or dysenteric diarrhœa, it is exceptional to fail to find such a history. It may be necessary to go back five, six, or even ten years, and the well-known slowness of the progress of a single abscess of the liver, before the symptoms become sufficiently marked to force themselves on the observation, makes it not unreasonable to look on such remote attacks of ulceration of the bowel as having been in reality the cause of the long-delayed manifestation of suppuration in the liver.

On post-mortem examination of fatal cases of tropical abscess of the liver, evidence of actual or previous ulceration of the bowel is present in a large proportion. Thus of one hundred and one European soldiers who died of hepatic abscess in India in 1899, actual ulceration of the large or small intestine was present in forty-six; intestinal scars in thirteen; and symptoms of dysentery had been present immediately before death in eight, though marked evidence of intestinal lesion was absent. This gives a distinct relationship to dysentery in 67 per cent of the cases. On the other hand, in fatal cases of dysentery alterations of the liver are present in about two-thirds of the number, and of these a fourth or a third show suppuration of that organ.

Again, the severity of the liver affection is in direct proportion to that of the intestinal lesion. Thus among the one hundred and one fatal cases of liver abscess among European troops in India in 1899, the abscess was single in thirty-five and multiple in sixty-six. In 77 per cent of the cases of single abscess there was no evidence of intestinal lesion, which was present in only 23 per cent, but 58 per cent of the cases of multiple abscess were associated with intestinal lesion, and in only 42 per cent was this absent. Among the forty-six fatal cases associated with ulceration of the bowel eight or 17 per cent were single abscesses, and thirty-eight or 83 per cent were multiple. In Sénégal, where dysentery assumes a virulent character, hepatic abscess is very common; whereas in the island of Réunion and in Algiers dysentery is of a less violent nature, and abscess of the liver is more rare.

While, therefore, the causal relationship of dysentery to hepatic abscess is borne out by statistics from every part of the globe, and is in consonance with the anatomical relationship of the bowel with the liver, and with the pathological risks which that relationship involves in septic ulcerations of the intestinal canal through the medium of the blood carried from the bowel to the liver by the portal system of veins, there remains a certain proportion of cases of tropical hepatic abscess in which there is an absence of distinct evidence of actual or previous intestinal lesion, and the question arises whether septic matter, or more properly organisms, capable of setting up sepsis in the liver, may not, under certain abnormal conditions of the bowel, be able to invade and pass through the intestinal mucous membrane without actual lesion. The analogy of typhoid fever without intestinal lesion, and the presence of coli bacilli in abscesses adjoining, but not involving the bowel, would favour the possibility of such an event; and the power of such organisms to

determine suppuration in the liver, when carried there by the portal vein, would then depend on their virulence, and the extent to which the resisting power of the liver was reduced temporarily or permanently by a blow, by chills, or by alcoholism. The frequent presence of the amœba coli in abscesses of the liver would not be incompatible with such a hypothesis. At the present time the question can only be put and not answered, because we do not have sufficiently precise data as to the exact character of the micro-organisms that exist in hepatic abscesses and their walls to say whether or not they are invariably such as have their usual habitat in the intestinal canal; but, granting the possibility of the invasion by alvine organisms of an unbroken surface of intestinal mucous membrane, the morbid condition which made it possible might be of such transient kind as to leave no trace when the bowel is examined sometime later, and thus the occurrence of liver abscess without evidence of intestinal lesion in a considerable proportion of cases would be accounted for.

Symptoms.—In a typical case of acute hepatic abscess the symptoms are clear and unequivocal in the advanced stages. The patient complains of pain, sometimes of an acute character, in the region of the liver and at the back or tip of the right shoulder, aggravated by movement and pressure. Decubitus is dorsal or right dorsal. Left lateral decubitus is prevented by dragging of the enlarged and inflamed organ on the suspensory ligament. This pain in the hepatic region is accompanied by a febrile condition of a continued or remittent character, the temperature rising in the evening to 101°-103° or more, and falling in the morning to about 100°. The face is flushed, the pulse full and bounding, the breathing quick and shallow, the tongue furred, the bowels active, and their contents bilious; the urine scanty and high-coloured, with or without indications of the presence of bile. The skin may be dry, or in the tropics it may be covered with profuse perspiration. There may be repeated rigors, the temperature running up to 104°, followed by exhausting sweats.

On uncovering the patient the hepatic region will be found to be distinctly fuller than the corresponding region of the left side, and more or less fixed. The intercostal spaces are widened and painful on pressure, and may be prominent if suppuration is near the surface; but under such circumstances, if adhesions have formed, this appearance may be obscured by local œdema. If the abscess be in the left lobe a distinct rounded tumour may be seen occupying the upper part of the epigastrium.

On palpation the lower margin of the liver will be felt some

inches below the right costal arch, rounded and very tender to pressure. The right rectus muscle is rigid and "on guard," preventing palpation of the liver in that part which it covers and protects. On percussion the upper margin of absolute dulness will be found higher than usual, it may be as high as the right nipple, and on marking the line on the surface it will be found to be curved upwards as it is traced from the sternum to the spine. The highest point reached by this line is an indication of the position of the abscess in the liver substance, if it be situated in the upper part, and pointing towards the diaphragm. The line of comparative dulness is also higher than normal, both actually and relatively, to the line of absolute dulness, and the parallelism of the two lines is lost, when the abscess is in the dome of the right lobe at some distance from the chest wall. The position of this divergence of the line of comparative from that of absolute dulness should be carefully noted as a guide to the situation of a deeply-placed abscess. When the abscess is more towards the anterior and lower margin of the liver it will cause a bulging downwards, which can be detected by palpation or careful percussion.

When the abscess is large and near the surface, either in the epigastric or costal regions, distinct indubitable fluctuation can often be detected by placing the points of the fingers of one hand in the widest intercostal spaces, and tapping with the other hand over the epigastrium, or *vice versâ*.

When the abscess is approaching the surface of the liver, and adhesions have not yet formed, a distinct to-and-fro friction murmur can sometimes be heard by means of the stethoscope. If audible in the epigastric region it is undoubtedly due to rubbing of the inflamed and dry peritoneal surfaces of the liver and parietes on each other; and it may be due to the same cause when heard in the costal region, but there it may be caused by pleurisy as a process preparatory to adhesion between the upper surface of the diaphragm and the costal pleura, when the abscess is pointing in that direction.

If the abscess be still small, and situated in the centre of the right lobe, the indications of its presence and position afforded by physical examination will not be so obvious, but the pain, and the tenderness on pressure, the enlargement, the rigidity of the right rectus muscle, the shivering and fever, ought to raise a suspicion strong enough to justify exploration, which should not be delayed, because in acute hepatic suppuration the progress and destruction of liver tissue are rapid, and an abscess the size of an orange may increase to that of a cocoa-nut in a few days.

In chronic abscess, which is also very common in the tropics, and is the only variety seen in temperate climates, the symptoms are less urgent, or they may be very obscure, and many of them entirely absent. Their obscurity is in proportion to the chronicity, and the clinical picture presented may therefore be very different from that of acute hepatic abscess.

The more usual course of such a case is as follows:—The patient, possibly some time, months or years, after an attack of dysentery, begins to feel languid and out of sorts, with an ill-defined uneasiness in the hepatic region and in the tip or back of the right shoulder. He loses flesh and appetite, and his countenance assumes a drawn, yellowish brown or muddy appearance. His bowels are irregular, with a general tendency to diarrhœa. He has a tendency to chilliness, or perhaps rigors, and his temperature is found to be above normal in the morning, and it rises to 101° or 102° in the evening. He takes quinine regularly, and in full doses, without any effect on the febrile symptoms. His head aches, his temper is irritable, he has disturbed nights, he cannot sleep comfortably on the left side, and he wakes bathed in perspiration. His appetite is capricious and poor.

On examination the liver will be found to be enlarged, both in its upper and lower limits, and if the abscess be not situated in the centre of the right lobe the upper line of percussion dulness will be found arched, most frequently in the axillary or nipple line, or it may be more posteriorly, so that the dulness in the scapular region may be as high as the eighth rib; or the lower margin will be found bulging downwards, or there may be a round resilient tumour below the xiphoid cartilage. Palpation elicits decided pain either at the point felt, or shooting upwards in the direction occupied by the abscess. The right rectus is rigid, or becomes so on any attempt being made to palpate the portion of liver which it covers. Breathing is shallow and thoracic, and the sufferer cannot be induced to take a deep inspiration while the fingers of the physician are held firmly in contact with the lower margin of the liver; or, if the enlargement be not so great, the action is checked directly the edge of the liver is brought into contact with the fingers. The hepatic region may appear fuller than it should be in a man so emaciated, and the intercostal spaces widened.

There is probably a history of repeated attacks of "pleurisy" low down on the right side, and if adhesions have taken place between the costal and diaphragmatic pleuræ, the mobility of the liver on inspiration and change of position will be distinctly lessened,

and a dragging sensation felt at the spot. If adhesion has not occurred, friction, peritoneal or pleural, may be audible, and the mobility of the liver will be lessened on inspiration by the fear of pain.

The urine is scanty, high coloured, of high specific gravity, and depositing urates copiously.

With the progress of the case emaciation increases, hectic symptoms, with night sweats, are constant, the enlargement of the liver becomes greater, and the febrile condition and general debility augment till the patient dies by exhaustion or some intercurrent complication, of which dysenteric diarrhœa is the most frequent; or the abscess may burst into some adjoining organ or cavity, either with rapid death if into the peritoneum or pericardium, or by exhaustion, or more or less slow recovery if into the lungs or gastro-intestinal canal.

Such are the symptoms and course of a well-marked case of chronic abscess of the liver; but if the abscess be small, centrally situated, or very slow in its advance, most of the indications of its presence may be entirely absent. The liver is not a very sensitive organ, and a small abscess, not in near proximity to its peritoneal covering, may give rise to little uneasiness and no enlargement. In such a case all the physical signs described above may be wanting, but pain can generally be elicited on deep inspiration by the fingers firmly placed below the costal arch. The febrile symptoms may be so slight as to escape notice, unless the temperature be taken systematically, and there may be considerable periods of complete freedom from fever. Rigors are by no means a constant symptom, and they are often, and in very chronic cases generally absent; they vary in frequency with the degree of fever present. Sweating, especially during sleep, is a very constant symptom, but it also may not occur. Emaciation is, however, a never-failing symptom, and, together with an anxious look, a muddy complexion, an unsatisfactory tongue, a variable appetite, depression and irritability, should always direct the attention to the liver in a patient who has resided in the tropics; and if he gives a history of previous dysentery, or of fever unaffected by quinine, and if at the same time distinct pain be felt on palpation of the lower border, sufficient grounds are present, in the absence of any other cause of ill-health, to suspect the existence of a chronic abscess of the liver, and to justify the employment of an exploring needle.

Diagnosis.—From what has just been said it is evident that the diagnosis of liver abscess must often be a matter of considerable

difficulty and doubt. There is probably no condition of equal gravity which is so frequently overlooked by practitioners unaccustomed to consider the probabilities of tropical disease; and as the diagnosis in these cases is of the most vital importance, it will be well to emphasise the difficulty by specific cases of mistaken diagnosis which have come under the writer's own observation. The conditions for which chronic abscess of the liver is most frequently mistaken are the following:—

Good health.—This is only possible when the abscess is small or of very slow evolution. In such cases there is no obvious enlargement of the liver, no pain, little or no abnormal temperature. In a recent case the various organs of the body were declared to be normal by a very competent observer, but suspicion was raised by the rigidity of the right rectus muscle when an attempt was made to palpate the left lobe of the liver, and on inquiry a history of an attack of dysentery eighteen months before was elicited. By placing the patient in a sitting posture on the edge of the bed, with the spine curved forward, the rigidity of the rectus was frustrated, and a small resilient tumour about the size of a Tangerine orange, very painful to the touch, could be distinctly felt on the under surface of the left lobe, a little to the right of the middle line. It was found by the aspirator to contain about a drachm of hepatic pus.

Dyspepsia.—If the abscess be pointing towards the under surface of the right lobe it may give little indication of its presence on physical examination; but the distension of the stomach by a meal, by causing pressure on the tumour, may give rise to pain after food, which may be mistaken for dyspepsia. A medical officer home from India consulted two physicians of his old hospital for symptoms of this character. They advised him to take plenty of out-of-door exercise, and recommended fox-hunting. He hunted two days without experiencing any harm, but while drawing on his boots on the third morning he felt something give way in the region of the liver, and he passed two pints of hepatic pus by the rectum. He made an excellent and rapid recovery. An abscess of that size ought to have given some sign of its presence on careful physical examination, or by an evening rise of temperature and a history of previous dysentery, and the patient should not have been exposed to the risks of the hunting-field with so large an abscess ready to burst into the hepatic flexure of the colon.

Malarial fever.—This is the error most frequently made in the tropics. Here there is distinct enlargement of the liver, with congestion and tenderness on pressure, and probably remittent fever of

a type which is common to both malarial fever and hepatic abscess. Enlargement of the spleen, which is the rule in malarial fever, is absent in some forms of remittent fever, and may be present, though it is usually absent in hepatic abscess uncomplicated with malaria. The presence of the plasmodium malariæ in the blood may be due to accidental infection during the progress of a case of hepatic suppuration. Under such circumstances resort should be had in the first instance to free dosage with quinine to remove the malarial element in the case; and then, if the temperature continue abnormal, careful examination of the liver will probably discover sufficient grounds for exploration if an abscess exists.

Influenza and its sequelæ.—Many cases of influenza are followed by a persistent but irregular daily rise of temperature, which may continue for weeks or months, the result of blood infection by one of the forms of micrococcus. There is often enlargement of the liver in these cases, and the resemblance between this condition and that caused by an abscess in the liver may be very close, and if, as sometimes happens, ulcerative endocarditis supervene on influenza, the infected vegetations may form the foci of septicæmic abscess in the liver. In a patient recently home from the tropics the condition is more likely to be one of tropical liver abscess from the beginning, with or without the complication of influenza, and to go on treating such a case as one of influenza will only lead to disaster. The mistake is one that can only be guarded against by remembering that every case of continued or remittent fever in a recent resident in the tropics, which resists quinine, may be due to an unsuspected abscess in the liver, and requires careful and repeated physical examination of that organ.

Typhoid fever and its relapses may be diagnosed when the symptoms are in reality due to hepatic abscess. The absence of Widal's reaction, the atypical course of the symptoms, and the growing urgency of the liver symptoms, ought to direct attention to the real cause of the illness. On the other hand, hepatic abscess may be the direct result of absorption from septic enteric ulceration of Peyer's patches, and thus become a sequela of typhoid fever.

Non-suppurative hepatitis and congestion, from whatever cause they may arise, especially if accompanied by irregular fever, as in chronic malarial poisoning, when all trace of parasitism has disappeared from the blood, are conditions which give rise to the greatest difficulty. The liver is considerably enlarged, and it may be distinctly irregular in outline at both upper and lower limits. There is considerable pain and tenderness on pressure, and at the

back or tip of the shoulder. There may be chilliness, or even shivering occasionally accompanying the daily afternoon or evening rise of temperature, and the profuse night sweats of febrile exhaustion. All the general symptoms which are the usual accompaniments of hepatic abscess may be present. In such cases, however, the spleen is usually greatly enlarged, reaching to or beyond the umbilicus, and there is the history of long-continued or often repeated attacks of a more or less distinctly malarial character.

The probability in such a case will be that there is no abscess in the liver, but this can never be asserted with any certainty, and exploration is not only justified, but imperative.

In such cases, and in other doubtful cases, some assistance will be afforded by a blood count, and especially by an enumeration of the leucocytes. The presence of an excess of the polynuclear forms would favour the existence of some active suppurative process; and if at the same time there were a marked diminution in the daily excretion of urea, the diagnosis of hepatic abscess would be greatly strengthened.

Empyema.—The general symptoms of hepatic abscess may closely resemble those of empyema, and an abscess situated in the upper and back part of the liver may raise the hepatic dulness as high as the eighth, or even seventh rib posteriorly, with absence of vocal fremitus and resonance, so that the physical signs may very closely simulate those of pus in the pleural cavity. The upper line of liver dulness is never so level as that caused by fluid in the pleura, nor does it vary so much with position, and it is continuous with the liver dulness in situations where there is no suspicion of fluid. Empyema, if in any quantity, will cause displacement of the heart, which an enlarged liver does not do in the same direction. The likelihood of making this mistake is much increased when there is a history of attacks of sharp "pleuritic" pain in the lower part of the chest, in the hepatic region, in reality due to intercurrent attacks of perihepatitis, or perhaps to true pleurisy preceding adhesions. Such a history is a very frequent one in hepatic abscess, and should direct attention to the liver rather than divert it to conditions of the pleural cavity. The crucial test is again the aspirating needle.

Pneumonia of the base of the right lung.—In many cases of hepatic abscess crepitation can be detected at the base of the right lung, but probably exclusively in those pointing upwards towards the diaphragm, as perforation of the diaphragm and discharge of liver pus through a bronchus is always preceded by consolidation and crepitation in the portion of the lung affected; and abscess of the

lung, as a sequel to the penetration of the pulmonary tissue by hepatic pus, is indistinguishable from ordinary abscess of the lung, except by the hepatic character of the pus expectorated and the history of the case.

Pleurisy.—Sharp attacks of pain of the nature of a stitch in the lower part of the chest on the right side are of very frequent occurrence in suppurative hepatitis, so much so that a history of repeated attacks of this kind in one who has lived in the tropics should always suggest a careful examination of the liver. These attacks are due to transient local perihepatitis lit up by an abscess near the surface of the liver, or to diaphragmatic or costal pleurisy in front of an abscess advancing towards the lung or side.

Rheumatism of the right shoulder.—Some patients, accustomed perhaps to a sense of fulness in the right side, which they put aside as "liver," may consult their physician on account of chronic rheumatism of a persistent or intermittent kind in the neighbourhood of the right shoulder or scapula, and neglect to mention their hepatic symptoms. It is a reflex pain, in which the hepatic branches of the phrenic are the afferents, and the local sensation of inflammation in the liver is conveyed through the fourth cervical, and felt in the branches of the cervical and brachial plexuses. This shoulder pain is present in from 15 to 20 per cent of cases of hepatic abscess. It is generally of a dull aching character, but is sometimes more acute. It may be confined to the top of the shoulder, or it may be felt in the region of the scapula or clavicle, or down the arm. It is always suggestive of liver trouble.

The following diseases may also be suspected when the case is really one of hepatic abscess, namely, softening gummata, suppurating hydatid subdiaphragmatic abscess, pyelitis, gall-stones, and inflammation of the gall-bladder, abscess of the abdominal or chest wall.

The symptoms on which the diagnosis must chiefly depend in difficult and obscure cases are therefore—

(*a*) Persistent and progressive emaciation without other obvious cause.

(*b*) An evening rise of temperature, persistent, and not due to active malarial or other infection, as proved by the microscopic and therapeutic tests.

(*c*) Occasional shivering, and more or less profuse sweats, especially during sleep.

(*d*) A history of previous intestinal ulceration.

(*e*) Uneasiness or pain in the hepatic region, especially on left

lateral decubitus, and a history of repeated "pleuritic" attacks low down on the right side.

(*f*) Pain in the tip or neighbourhood of the right shoulder.

(*g*) Rigidity of the rectus muscle when an attempt is made to examine the liver.

(*h*) Pain on palpating the liver below the costal arch, or on percussion over its exposed part.

(*i*) Enlargement of the organ, especially if the enlargement shows local irregularity in outline as ascertained by percussion.

To these, as less constant symptoms, may be added pain on deglutition or after food, a reflex cough, ascites, and œdema of the hands and feet.

The existence of these, or of a considerable number of these signs and symptoms, demands the use of the exploring needle.

Exploration is both therapeutic and diagnostic in its uses. It is a common experience to find that non-suppurative hepatitis undergoes marked improvement after exploration with a pneumatic aspirator used with the necessary aseptic precautions. It seems to act as a local depletant with the best results. The proceeding, when indicated, is also safe, and if no pus be found, it is not only harmless, but may be beneficial. The use of the aspirating needle should, therefore, never be delayed when there are grounds for suspecting the possibility of the existence of an abscess for fear of doing any mischief to the inflamed organ if no pus be found.

Exploration, when indicated, must be thorough. If the needle be aseptic, and not more than four inches long, it may be made to search the liver substance in many directions without risk. The site of the first puncture will be in the most likely position, as indicated by physical examination. A needle thrust in to its full depth may pass through a small abscess into healthy tissue beyond it. It should, therefore, be withdrawn slowly while still communicating with the exhaust bottle. The depth of the needle on the appearance of pus should be noted.

If no pus appear on the slow withdrawal of the needle, it should be thrust in again in a different direction through the same puncture, as in its first transit it may have missed the abscess cavity by a fraction of an inch, regard being had to the concavity of the normal under surface of the liver in giving it a new direction. If no pus be found the process may be repeated in two other positions chosen according to the extent of liver dulness present. It may be necessary to explore in seven or eight directions at one sitting.

A large and deep incision in the skin is not necessary in order to explore the liver. An incision through the thickness of the skin

half an inch in length is sufficient to allow the entry of the needle without undue pressure.

Hepatic pus is peculiar and characteristic. It is thick in consistency, and of a reddish colour, closely resembling anchovy sauce. It is so thick that, in exploring, a needle of considerable calibre must be used. The redness, which varies in degree inversely with the chronicity of the case, is due to the presence of blood. The contents of a hepatic abscess are at first almost invariably sterile, a fact which enables surgeons to deal with the case with considerable freedom. The living organisms associated with the process, the amœba coli, and the other organisms, probably originally derived from the intestinal canal, are not at first found in the pus cavity, but are hidden in the spongy, finger-like processes which constitute its confining walls, and only appear in the discharges after twenty-four or forty-eight hours, or longer. In very chronic cases where there is a living "pyogenic" membrane the pus may still be aseptic, but it loses its characteristic appearance.

Terminations.—If left entirely without treatment of any kind, death occurs in three out of four cases, and in one-half of them without the suppuration having transgressed the limits of the liver. Among those in which it had passed beyond the limits of the liver, Rouis found that in six it had begun to affect adjacent organs, in twenty-six it had discharged into neighbouring closed cavities, fourteen times into the peritoneum, eleven times into the right pleura, and once into the pericardium; and in thirty it had found an issue outside the economy thus :—

By the lungs and bronchi	15
By bursting into the stomach . . .	5
„ „ colon . . .	3
„ „ duodenum . . .	1
„ „ gall-bladder . .	1
„ „ biliary ducts . .	1
„ „ pleura . . .	2
By an opening on the surface . . .	2

Of thirty-nine cases of spontaneous recovery all had succeeded in obtaining a direct passage to the exterior or to a mucous membrane. Thus there were :—

Through the thoracic or abdominal wall .	17 cases
Through bronchi	15 „
Into the stomach	3 „
Into the colon	4 „

Of the seventeen cases which found an opening through the skin, the opening was in the epigastrium thirteen times, in one of the lower intercostal spaces three times, and at the umbilicus once.

In actual practice, that is in cases under treatment, spontaneous rupture through the abdomino-thoracic wall is rare, for the reason that the surgeon interferes before it can become completed by natural means, and rupture through the lung is the most frequent occurrence, because abscesses situated deeply towards the upper curvature are most liable to escape detection, and these find their most natural exit upwards through the lungs. Similarly, rupture into the hepatic flexure of the colon is more often met with than rupture into the stomach, because an abscess pointing towards the stomach has more obvious symptoms than one situated in the right lobe in the neighbourhood of the hepatic flexure. Sometimes the pus from a hepatic abscess may seek an opening a long way from the liver, especially when it opens posteriorly and is prevented from reaching the surface by the dense fasciæ of the lumbar region. An abscess over the upper part of the sacrum may thus be in direct communication with a suppurating cavity in the liver.

Rupture into the peritoneum, into the pericardium, or into a blood-vessel, is rapidly or immediately fatal.

Rupture into the stomach is signalised by vomiting of hepatic pus, and relief to the previous symptoms. After rupture into the transverse colon there is similar relief, and hepatic pus may, if looked for, be seen in the evacuations, which are usually diarrhœic. This is one of the most favourable natural terminations of hepatic abscess, but sometimes if the opening into the bowel be large, flatus and fæcal matter find their way into the abscess cavity with very disastrous results.

Rupture into the lung occurs when the abscess is situated in the upper and back part of the liver, this being then the direction of least resistance. It is usually preceded by fairly well marked evidence of pneumonic consolidation, and the true nature of the case may not be suspected till the patient begins to expectorate hepatic pus. The quantity expectorated will depend on the size of the abscess, and the freedom of the communication with the bronchus. Occasionally the lungs are suddenly inundated with pus, and suffocation may occur, but more usually the quantity with each cough is small, though it may amount to eight to twelve ounces in twenty-four hours. There is generally relief, especially to the febrile symptoms, when this exit to the pus is accomplished; but very often the communication is small, and perhaps tortuous, and the discharge is not

continuous. There are, therefore, very often periods of apyrexia with free expectoration, followed by a gradual recrudescence of the febrile symptoms, with possibly rigors, till the communication is re-established. Such cases do badly, and surgical interference becomes necessary to facilitate the discharge either by an operation on the lung, or by cutting down on the hepatic abscess, if by any means its position and extent can be ascertained. In many cases of long duration the hepatic lesion is, however, reduced to a mere cup-shaped depression on the upper surface of the liver, and the condition is then practically one of pulmonary abscess, and can only be treated as such. These cases which terminate by rupture into the lung are on the whole favourable, but may be of very protracted course, extending into months, and even years, of impaired and precarious health. In favourable cases the daily amount of expectorated pus gradually lessens, and finally ceases with complete recovery. If this does not happen the deduction is, either that the passage through the lung does not afford sufficient free drainage, or that the case is one of multiple abscess.

Rupture into the pleural cavity may occur on either side, but is not a frequent accident on account of adhesions forming in front of the advancing inflammation. It may be followed by shock if the quantity of pus discharged into the pleura be large, with pain and dyspnœa, and subsequent signs and symptoms of empyema.

Prognosis.—Prognosis is always grave. Tomes estimated the mortality of unassisted cases at 90 per cent, Rouis found that two hundred and three such cases gave a death-rate of 80 per cent. In the tropics, including a large number of acute cases, the mortality, even under skilled treatment, is very high, amounting among European soldiers in India in 1899 to 49 per cent. Among the more chronic cases which come under observation in temperate climates the proportion of deaths is much lower, and probably does not exceed 10 per cent.

While multiple abscess of the liver may be said to be invariably fatal, an analysis of the figures given by the returns of the Government of India shows that in that country the death-rate from single abscess under surgical treatment, inclusive of many acute cases, is under 30 per cent. A chronic single abscess which has reached a temperate climate, if opened and freely drained, and if it has not attained to large dimensions, with corresponding destruction of liver tissue, may be regarded as offering a very reasonable hope of recovery.

PERIHEPATITIS

Definition.—Inflammation of the peritoneal covering of the liver.

Perihepatitis may be acute or chronic.

Acute perihepatitis occurs as a complication or extension of acute inflammation of the liver substance, of the peritoneum, or, through the diaphragm, of acute pleurisy. It may be general, as a part of general peritonitis; or partial, confined to the upper surface, as the result of pleurisy; or in patches, when it is an extension of suppurative inflammation below the surface of the liver. It is of frequent occurrence in abscess of the liver; and in all cases in which adhesions are forming in front of a hepatic abscess it is an essential part of the process by which the liver is glued to an adjacent organ prior to spontaneous evacuation of the pus.

The **symptoms** under such circumstances are, in addition to those of the condition which is already present, the supervention of pain in the hepatic region, sometimes of a very acute character, requiring the use of morphia injections for its relief, but usually lasting only a few days; quick, shallow, thoracic breathing, fixation of the lower ribs on the right side, and great tenderness on palpation or percussion of the exposed part of the liver. On auscultation a distinct friction murmur can often be distinctly heard. When such attacks occur in the progress of a hepatic abscess they are often regarded as indicative of diaphragmatic or costal pleurisy, but a succession of such attacks should always raise a suspicion of liver trouble in a patient who has been exposed to climatic or other causes of hepatic abscess.

This partial acute perihepatitis rarely spreads, and generally ends in adhesion of the opposed visceral and parietal surfaces.

Chronic perihepatitis may be general or local. *Local* chronic perihepatitis is almost always due to previous disease existing in the liver or adjoining organs, and differs from local acute perihepatitis in the firm connection of the inflamed surface with the subjacent liver tissue, so that it cannot readily be peeled off, and it may have a depressed or pitted appearance. It is seen in cancerous or syphilitic tumours of the liver, in tuberculous disease, round the gall-bladder in biliary calculus, and in chronic abscess near the surface. It may occur as a result of disease in neighbouring organs, especially in gastric ulcer, which may thus become adherent to the

liver, and it is common in passive congestion, resulting from obstruction to the flow of the blood in the lungs and heart.

It gives rise to few symptoms beyond local pain and uneasiness, and is not usually detected during life.

General chronic perihepatitis is, according to the observations of Dr. Hale White, seldom, if ever, of local origin or causation. It is, as a rule, a part of a general chronic peritonitis, and in a large majority of cases is associated with, if not caused by, interstitial nephritis or syphilitic disease. The general and non-local origin of this form of perihepatitis is shown by the frequent implication of the spleen in an exactly similar form of capsulitis.

In this form of disease the liver is enclosed in a thick, opaque, white coat, which can be peeled off the smooth surface below. A similar appearance is given to the spleen, and in many cases a layer of the same kind can be separated from the general peritoneum; in others the general peritonitis is of the thickened and opaque kind, common in chronic inflammation, and is not associated with tubercle or other growth of the peritoneum.

The liver substance is but little altered in typical cases. It is seldom cirrhotic, but it may be lardaceous in syphilitic cases, and present the nutmeg appearance of passive congestion if the lungs and heart be affected as a result of the kidney affection. The anterior margin of the liver is often turned backwards and adherent to the upper surface.

Symptoms.—There are few symptoms referable to the liver in general chronic perihepatitis. Jaundice is rare, because the exudation exerts little or no pressure on the bile ducts at the hepatic fissure. Ascites is, however, a constant symptom, not due, according to Dr. Hale White, to constriction of the portal veins, but to the general peritonitis, of which the perihepatitis is only a part. The ascitic fluid is clear, and reaccumulates repeatedly, so that many tappings may be necessary. The urine nearly always contains albumin, and there are the other indications of chronic interstitial nephritis, which is a constant factor in the condition and leads in many cases to heart failure.

Diagnosis.—The clinical difficulty which these cases present is that of distinguishing between general chronic perihepatitis and ascites, due to cirrhosis of the liver. If it be borne in mind that the former is a part of a general peritonitis, and is nearly constantly associated with interstitial nephritis, the diagnosis becomes less difficult. In the first place, evidences of chronic peritonitis must be looked for. Thickening of the peritoneum will be made out in

some part of the abdomen, and especially thickening and puckering of the omentum, giving rise to a feeling of induration lying transversely across the abdomen above the umbilicus. For the same reason the bowels are generally retracted towards the spinal column, and do not float in the ascitic fluid so freely as when peritonitis is absent, and the dulness on percussion is therefore more general and rises higher. When there are marked signs of interstitial nephritis the presumption is in favour of perihepatitis.

On the other hand, interstitial nephritis is often associated with cirrhosis; but here there are marked changes in the volume of the liver, and in its texture on palpation; jaundice is a frequent complication, and there are no indications of chronic peritonitis.

Cirrhosis is five times more frequent than chronic general perihepatitis.

Prognosis.—The condition is progressive, and always fatal.

Treatment.—Beyond relief by repeated paracentesis little can be done to mitigate the progress of the disease.

A. Crombie.

DEGENERATIONS OF THE LIVER

Fatty Liver

Symptoms.—As fat is a normal constituent of the liver it is not surprising that a moderate excess of fat will not excite attention by subjective symptoms or objective signs. But when the fatty liver assumes large dimensions, it may from its mere bulk give rise to feelings of discomfort, more pronounced when the patient lies on his left side. The accurate determination of an enlarged fatty liver is often attended with some difficulty, as the owner is frequently the subject of general obesity; but, under favourable circumstances, the area of hepatic dulness will be found to be much increased, and the margin much lower than normal. If palpable, the anterior border will be felt to be rounded, with the fissure in the left epigastric or umbilical region. The general surface is smooth, and there is usually no tenderness experienced when handling it. As a rule there is no jaundice, but in rare cases where the liver is enormously enlarged there may be towards the end both jaundice and tenderness.

Where a fatty liver is part of a general obesity, the presence of the enlarged organ may be associated with symptoms due to upward pressure on the diaphragm, such as dyspnœa and palpitation, which

are often aggravated by distension of the stomach by food or flatulence. It is difficult, however, to indicate accurately what exact relation the enlarged fatty liver has to the general symptoms in cases of obesity, as the deposit of adipose tissue is so universal, and in the case of the heart is often sufficient of itself to produce the breathlessness. With regard to the torpidity and irregularity of the bowels in some of these cases, the enormous deposits in the subserous tissue of the abdomen are sufficient, from a mechanical point of view alone, to account for these. It is also difficult to draw the line between the accumulation of fat, such as occurs in obesity, where it may be hereditary or racial, or happening after the change of life, or where it is the outcome of indolent habits and luxurious living, and cases where it is associated with disease and toxic infections, for some of the causes influencing the determination of fat in the liver are common to most classes. At any rate, it may be asserted safely that there is a large fatty liver the result of the prolonged use of alcohol, and this without being associated with cirrhosis. In all cases of cirrhosis a fatty degeneration of the liver cells is a feature, and in some it is a very pronounced one, so much so as almost to warrant the use of the term fatty cirrhosis. In any wasting disease it may frequently be observed, such as cancer, chronic suppuration, and pulmonary phthisis. In consumption it is very frequent, and is observed more often in females than in males. All these examples have one factor in common, that of deficient oxidation, though possibly some of the fat may be deposited by accumulation when the patient has been treated with cod-liver oil and much oleaginous food. Eminent examples of fatty degeneration due to bacterial, metallic, and possibly autogenous toxins, are to be found in phosphorus poisoning, acute yellow atrophy, pneumonia, yellow and other fevers.

A **diagnosis** of fatty liver may be arrived at by ascertaining that the general shape of the organ is maintained; that it is smooth, and, though firm, not indurated; pain, jaundice, and ascites are usually absent. The associated conditions afford considerable assistance in arriving at a conclusion, but in many instances we must be satisfied with a probable inference, the result of experience.

As the fatty liver is merely the associate of a general condition, **treatment** must be directed to the alleviation or removal of the disorder, such as phthisis, cirrhosis, marasmic and plethoric states. It may, however, be convenient to recall here the lines of treatment suitable for dealing with the fatty liver of obesity.

If the condition be acquired from luxurious living, high feeding,

and indolent habits, much may be done; but it is a great question if permanent and real good will ensue from trying to reduce a natural and hereditary adipose condition of body. The attempt to extract fat from an abnormally adipose person is often about as easy as endeavouring to reduce the fibrosis in a cirrhotic liver, but some cases are amenable to treatment, and the diminution of weight is not followed by ill effects to the system. The method is, in its essential features, a combination of the regimen ordered for diabetics and the practice adopted by athletes and jockeys for the reduction of their weight. Of course no exact set of rules to be observed in all cases can be given, as each case must be treated on its own merits, and under the careful supervision of a competent adviser. Thus some do better when taking exercise and sweating, others when the diet receives most attention. Turkish baths and general massage are efficient auxiliaries, and it is a cardinal rule that the daily quantity of liquid should be strictly limited. Attempts to reduce the weight with too great rapidity, by taking too little food, by too severe and prolonged exercise, and by the use of drugs and quack nostrums, have been followed by disastrous consequences (see article "Obesity," Vol. II. p. 168).

Lardaceous Liver

Symptoms.—Like fatty degeneration the lardaceous change in the liver is a concomitant phenomenon of those underlying conditions to which it owes origin, and as it gives rise to but slight indications of hepatic derangement, its detection in the early stages is more a matter of surmise than certainty. The invariable tendency of the degeneration is to produce a uniform enlargement of the viscus. Lardaceous livers are often not only bulky, but heavy, and may weigh as much as 8 lbs. or more. The organ becomes uniformly enlarged, and when the increase of size is great there is obvious bulging of the upper part of the abdomen, more marked on the right side. The organ feels firm, even hard to the touch; its surface is smooth, and its edge distinctly rounded. It is not tender on being handled, nor is it painful, though it may give rise to some discomfort from its size, and from pressing unduly on adjacent structures. The degeneration does not give rise to marked special symptoms, but deficient secretion of bile, with light-coloured, even clayey stools, are often present, and this deficiency in its turn reacts on the general nutrition by impeding intestinal digestion and absorption (see Vol. II. p. 99).

The **diagnosis** from other enlargements of the liver depends mainly on the associated and underlying conditions. The most common of these are prolonged suppuration from bone disease, empyemata, cavities in tuberculous lungs, especially when the source of the pus is deep-seated and does not allow of good drainage. Another common cause is syphilis, congenital or acquired, whether attended with suppuration or not. Less frequent sources of the degeneration are malaria and new growths.

Further assistance is derived from enlargement of the spleen, gastro-intestinal symptoms, and evidence of the existence of lardaceous kidney, as indicated by much albumin in the urine and the presence of waxy casts.

A **prognosis** *qua* lardaceous liver can scarcely be required, for any symptoms it may occasion are overshadowed by the conditions giving rise to it. If these disappear, so too will the lardaceous substance, a fact which rather supports the view that the albuminoid material is merely deposited in the affected parts. Should, however, the symptoms of the exciting condition ingravesce, then no doubt the lardaceous change in the liver will co-operate, and thus make the prognosis graver.

The **treatment** of lardaceous degeneration must, in the first place, be directed to the removal of the exciting cause, *e.g.* by draining cavities, removal of dead bone, and by the administration of antisyphilitic remedies. No treatment can hope to be successful unless this fundamental proposition be adhered to. Tuberculous intrapulmonary cavities stand, however, on a somewhat different footing from an empyema sac, and it is usually advisable not to meddle with them. Vomiting and diarrhœa should be checked at once, for they are both distressing and weakening. For the former hydrocyanic acid, ammonium bromide, or cocaine hydrochloride will be useful; and for the latter astringent preparations, such as compound kino powder, chalk and opium powder, Dover's powder, opium, catechu, or lead and opium pill. As these lardaceous conditions are attended with anæmia, frequently profound, and not seldom by dropsies, the maintenance of the general health and strength is of prime importance. This must be done by suitable and nourishing diet, and by the exhibition of tonic and absorbent remedies, such as iron, syrup of the iodide of iron, hypophosphites, cod-liver oil, or ammonium chloride. Sea air and change of climate have often aided in ameliorating the condition.

ACUTE YELLOW ATROPHY

Symptoms.—The onset of this malady is usually gradual, especially in children, there being for the first few days or even for a week or two little to distinguish it from simple or catarrhal jaundice. The jaundice may be accompanied or even preceded by pyrexia, vomiting, and pain and tenderness over the site of the liver. Frequently, however, the first symptom to attract attention is jaundice, followed after the lapse of a variable interval by gastro-intestinal and nervous symptoms, which may develop with remarkable suddenness and intensity. Though the general outlines of the clinical picture are much the same, the details may exhibit differences both in the first and final stages. In some there is malaise, headache, irritability, followed by drowsiness deepening into coma, with stertorous breathing and general flaccidity of the muscles. In others wild delirium and convulsions immediately precede the comatose stage. In others, where jaundice and irritability have been the principal features, there may be a sudden lapse into unconsciousness, followed by convulsions till death. In all the jaundice goes on increasing, and the vomit, which at first is usually bilious, becomes towards the last coffee-ground in character. The tongue is furred and often covered with sordes. There is usually constipation; the stools are deficient in bile, may be clay-coloured, and towards the last tarry from the presence of blood. The urine is bile-stained and may contain leucin and tyrosin. In most cases the urea is deficient, but in some is in excess of the normal; in some the presence of albumin and casts has been noted. The cardiac action is feeble, rapid, and often irregular.

The disease mostly runs an afebrile course, but in some instances there is pyrexia, and certainly towards the end the temperature rises even as high as 107°.

In some the skin is hyperæsthetic, the patient screaming when touched. Hæmorrhages into the skin and from the mucosas are frequent.

Pregnant women usually abort. In children the onset is more insidious than in adults.

Examination of the abdomen during the progress of the disease shows that the liver dulness decreases in extent, until finally it may disappear altogether. In the early stage the liver is either normal or even increased in size. There is usually tenderness in the epigastric and right hypochondriac regions.

The duration of the disease is very uncertain, but when the gastro-intestinal become marked, and the nervous symptoms declare themselves, it is only a question of a few days.

Diagnosis.—In the early stage there may be nothing to distinguish acute yellow atrophy from a simple jaundice, and it is only when the nervous and gastric symptoms arrive, coupled with a knowledge that the liver dulness is continually decreasing, that a diagnosis becomes possible. The disease for which it is most likely to be mistaken is phosphorus poisoning, but from this it may be discriminated by the greater size and tenderness of the liver, by the epigastric burning pain, by the constant vomiting which usually comes on soon after swallowing the poison, by the garlicky odour of the breath, and by the luminosity of the vomit when inspected in the dark. Very often these symptoms partially subside for awhile and are then followed by jaundice, with gastro-intestinal and nervous phenomena. In acute yellow atrophy the jaundice is prodromal, and may be associated with few or no marked symptoms. The clinical record is marked by no drop; the symptoms are always ingravescent. The heart and kidneys are much less affected, and leucin and tyrosin are more frequently found in the urine than in phosphorus poisoning.

The **prognosis** is always extremely grave, and some authorities express doubt as to the possibility of recovery. Yet cases of recovery have been reported, and a few are stated to have succumbed to a second attack. Until more is known of the exciting etiological factor or factors, it would be unwise to express a decided opinion either way.

Treatment.—As the etiology of this malady is quite unknown, and as all that pathology can do at present is to point out the morbid appearances, appearances which may be described as a bacterial toxin-necrosis, the treatment must be purely empirical and conventional, but assuming the bacterial origin, the line to be adopted is symptomatic and antiseptic. In the early stage it will be advisable to clear the bowel, and for this purpose calomel or gray powder is theoretically indicated, but saline or cholagogue purgatives apparently effect the desired end equally well. Diaphoresis should be promoted by means of hot air baths or by sudorifics, such as pilocarpin and acetate of ammonia. For the vomiting, hydrocyanic acid and soda, morphia, or ammonium bromide should be tried, and for the hæmorrhage, ice, morphia, tannic acid, hazelin, ergotin, or adrenalin. Too much, however, must not be expected from the foregoing, and until a remedy capable of neutralising the

toxin without, at the same time, damaging the organism, has been discovered, we must be content with attempts to alleviate the symptoms.

NEW GROWTHS OF THE LIVER

Symptoms.—At their inception new growths in the liver, whether primary or secondary, rarely give rise to symptoms of hepatic derangement, and the detection of their presence is usually contemporaneous with symptoms and signs pointing to enlargement of the viscus and irregularity of its surface, together with implication of the bile ducts and of the portal vein. Probably quite 95 per cent of malignant growths in the liver are secondary from metastasis or by continuity, the primary source being usually some part of the alimentary canal, most often the stomach. Secondary deposits are not infrequently latent, and their discovery may only date from the post-mortem examination, but often enough malignant growths in the liver are easily detected when they have attained certain dimensions, and when the presence of cancer elsewhere is known or suspected.

When cancerous affections of the liver become diagnosable the organ is nearly always enlarged, its outlines irregular, and the surface often nodose or bossy. On inspection a bulging in the right hypochondriac and epigastric regions may be evident. By percussion, palpation, and cyrtometric measurement, the enlargement of the viscus and the abnormal contour of trunk may be demonstrated. Usually there is tenderness on manipulation, and very occasionally, owing to the central liquefaction of a large mass, an obscure sense of fluctuation may be experienced. The spleen is very seldom enlarged. There may be more or less ascites, and this, associated with distension of the bowel, may tend to mask the condition of the liver. In the greater proportion of cases there is jaundice, certainly so towards the end, due to pressure on the main bile ducts by the growth or by enlarged glands in the portal fissure. The discoloration may be variable in degree and even in distribution, but when once it sets in it goes on increasing and often becomes intense. Very frequently pain is a significant symptom; its specific characteristic is that it radiates in all directions from the organ to the spine, thorax, and abdomen; it may be dull, heavy, continuous, boring or fitful, shooting and excruciating, and often worse at night.

As the condition progresses, and the liver keeps on enlarging,

the patient not only gets weaker and more emaciated, but certain symptoms develop or become more marked. Owing to its bulk it may interfere with the diaphragm, the lung, and the heart. It may press on the cava behind. By extension into the fissure the portal circulation may be interfered with and ascites become extensive, and a collateral circulation along the anterior abdominal wall may be established. Symptoms of gastro-intestinal irritation and congestion may become apparent and reveal themselves by nausea, vomiting, and hæmorrhage. Pyrexia of an irregular type and usually of moderate degree may be present. The jaundice is often accompanied by pruritus, often intense, by petechial extravasations into the skin, and hæmorrhages from the mucosas. There is deficiency or absence of bile from the stools, and its presence in the urine. Towards the last there are often nervous symptoms, delirium, coma, due to hepatic inadequacy.

The symptoms of primary carcinoma are extremely vague, but in the few cases I have seen the history has been progressive weakness and emaciation, accompanied by considerable pain in and radiating from the liver. The pain is worse at night. After a lapse of some months attention is drawn to the liver, and examination then shows that its size and shape are abnormal. Still later jaundice supervenes, and not till this appears do gastro-intestinal symptoms occur. The patients died of asthenia or cholæmia.

Sarcoma is almost invariably secondary, and nearly always subsequent to melanotic sarcoma of the choroid. The infiltrated organ usually becomes extremely bulky, and the symptoms do not materially differ from those of carcinoma.

Angiomata and adenomata are more or less pathological curiosities.

Diagnosis.—The recognition of the malady is not always apparent, since hepatic cancer may occur without nodulation or even enlargement of the organ, without pain or tenderness, and not at all infrequently without jaundice or ascites. When the disease is well established a diagnosis may often easily be made, and in the main will depend on the size and shape of the liver and the associated conditions of emaciation, pain, and jaundice, while much assistance may be derived from the knowledge of disease elsewhere, whether malignant or not. In the early stages a positive diagnosis should be suspended, but the age, say forty to fifty, a pallid and sallow aspect, localised pain and tenderness, and progressive emaciation, as well as the absence of symptoms pointing elsewhere, may be of service.

When the liver is enlarged and irregular in shape the hepatic conditions, which may be confused with malignancy, are mainly syphilis, hydatids, and hypertrophic cirrhosis. In syphilis there are the history, the age, the condition of the skin and mucosas, etc., and the much less rapid progress of the symptoms, to assist in arriving at a conclusion, which will be supported by the effect of treatment with antisyphilitic remedies.

In the large cirrhotic liver there will be a history of alcohol, extreme irregularity of the hepatic surface will be absent or much less marked, there will be enlargement of the spleen, and the jaundice will be less intense and less progressive.

Hydatids cause much increase in bulk and bossiness of surface, and may occasionally be accompanied by the presence of cysts in the peritoneum which impart to the abdomen an irregular appearance. Taken by themselves these signs might and have been deemed indicative of cancer of the liver with secondary deposits, but the history alone is almost sufficient to differentiate, for hydatid cysts increase in size slowly, are almost painless, and only cause discomfort by their size and proximity to adjacent parts, or produce symptoms by their rupture or suppuration, and unless the patient be afflicted with another disorder there are no concomitant general symptoms, no cachexia, no debility, no wasting.

The liver is much increased in size in lardaceous degeneration, in leukæmia, and lymphadenoma, but the associated conditions are so marked and distinct that it is hardly necessary to do more than point them out.

Much importance has been attached to an absence of splenic enlargement as a diagnostic sign of cancer, especially since it may occur with many of the other hepatic affections for which cancer may be mistaken; but in the consideration of this sign care must be taken not to confound an enlarged left lobe of the liver or a gastric growth with the spleen; and, on the other hand, splenic cancer is not absolutely unknown.

Prognosis.—When the diagnosis of cancer is made it is practically equivalent to signing the death certificate, though the exact date of demise can only be approximately indicated. As a rule the interval between the declared symptoms and the end is not more than twelve months, mostly less, but cases are reported which have lived for at least two years after a definite diagnosis has been made.

Treatment.—Of course no treatment is of any ultimate avail, but the patient's sufferings may be much alleviated by palliative measures directed to the symptoms. For the pain morphia should be given

hypodermically. Opium is most suitably administered by suppository. Some patients find relief from local applications, *e.g.* fomentations, stupes, leeches. Often the stomach is primarily or indirectly affected, and gastric symptoms require relief; they may be severe enough to necessitate rectal feeding, but usually nausea and vomiting may be controlled by hydrocyanic acid and soda, ice, or morphia, while the dyspepsia may be alleviated by ox or pig bile, the liquor pepsinæ, or liquor pancreaticus. The alimentation requires considerable attention. Only small quantities of food should be given at a time, and then should be easily digestible or predigested. Sometimes a particular substance, such as panopepton, may be retained and all others rejected. It is frequently wise to accede to the wishes of the patient in regard to articles of diet, for as he is stricken with a mortal disease it may be advisable to satisfy him. The itching attendant on the jaundice may be mitigated by sponging the surface with warm washes containing carbolic acid, hydrocyanic acid, or cocaine. The presence of fluid in the peritoneal sac is best disregarded, unless its accumulation very materially interferes with the action of the thoracic and other viscera.

CIRRHOSIS OF THE LIVER

Symptoms.—The early symptoms of cirrhosis of the liver are usually shrouded in obscurity, chiefly owing to the fact that the patient's complaints are not definitely hepatic, though it must be admitted that many examples of the early stages should be included in that vague collection of symptoms known to the laity and the profession as "the liver being out of order." In some there have been, from time to time, symptoms of chronic dyspepsia, such as flatulence, irregularity of the bowels, discomfort from distension or from a feeling of weight in the upper part of the abdomen, nausea, anorexia, and a bitter taste in the mouth.

As time goes on these symptoms become ingravescent, and the attacks are more frequent. The patient complains that he is out of sorts and that he suffers from biliousness, and often ascribes his condition of debility to being unable to take sufficient nourishment owing to want of appetite. In others the symptoms are plainly those of chronic alcoholism, the free use of which the patient may even admit. He complains of want of appetite, morning nausea, headache, insomnia, nervousness, that he "doesn't feel up to the mark." "He wants a tonic, and just something to regulate

his bowels," for they are very uncertain in their action, there being sometimes constipation, sometimes the reverse. His breath is foul, his tongue furred, and his conjunctivæ injected.

Two types of the cirrhotic habit may be discriminated, the one spare and thin, the other bloated and obese. In both the small veins of the cheeks are often characteristically evident, and while the complexion of the former is more often marked by pallor, the latter has a sallow, muddy, often yellowish tinge of skin. Speaking generally, these two types will be found to correspond roughly with the condition of the liver found later, viz. the small and large cirrhotic livers. But the gradations between the two, *i.e.* the mixed types, are just as numerous as those between the size and weight of the livers, and have some relation to the ascites and jaundice and the amount of fat in the liver. In both there is almost invariably a history of alcohol; and in connection therewith, it should always be borne well in mind that some persons may imbibe huge quantities of all kinds of alcohol without any marked effect on any part of the organism, while in others minute doses are sufficient to produce very deleterious and often lasting results. After a variable length of time, according to the idiosyncrasy of the patient towards the alcoholic toxin, other symptoms make their appearance. Piles begin to render existence uncomfortable; they bleed; get inflamed. Bleeding from the nose, sometimes very copious, issues warning notes from time to time that the constitution is being undermined. But the patient takes little heed, and as the feelings of general discomfort are often temporarily alleviated, the loss of blood is even regarded as an effort of nature to relieve the system. Years pass on, and in the interval some rarely go to bed sober, and it may be quite ten years before any noticeable enlargement of the liver can be detected, and even another ten before the classical symptoms of cirrhosis declare themselves.

Possibly the first event to startle the patient is the occurrence of hæmatemesis, followed by melæna. The latter is often unnoticed, but occurs more frequently than is suspected. The amount of blood vomited may be large, and recurrences may be frequent. The source of the hæmatemesis may be rupture of œsophageal varices, or from engorged gastric veins, more rarely from actual ulceration; while the melæna may, in addition to the foregoing sources, take origin in the congested condition of the intestinal mucosa. The occurrence of hæmatemesis is usually soon followed by other symptoms due to implication of the portal circulation, or of the biliary canaliculi, or some associated condition, such as degenera-

tion of the heart muscle and Bright's disease. Hence ascites, anasarca, albuminuria, jaundice, palpitation, and breathlessness. The general condition may be further aggravated by the presence of some concurrent lowering disorder, such as tuberculosis.

During the stage preliminary to the advent of symptoms indicating that the hepatic gland is no longer adequate to perform its functions, the organism has attempted to obviate the obstruction arising from the formation of fibrous tissue along the portal canals by means of a compensatory collateral circulation. Possibly the pseudo-ducts, ineffective though they be, may also represent an attempt of nature to provide a new system of biliary canaliculi. During life the evidence of a collateral circulation is sometimes shown by the obvious enlargement of veins coursing along the abdominal wall, and by the presence of hæmorrhoids. Post-mortem, however, the direction of the compensatory circulation is less obvious, though sometimes it may be demonstrated with accuracy. Thus the varicosity of the œsophageal and rectal veins indicates a junction of the upper and lower ends of the portal with the systemic circulation, whilst the radicles of the portal vein intervening between these two points may find a junction with the retro-peritoneal. Another important path of communication is formed by parumbilical vessels running along the round ligament to the umbilicus, about which they occasionally form a well-marked network before they pass upwards along the anterior abdominal wall. Possibly also new venous channels are formed in the adhesions which sometimes unite the liver to the diaphragm and the wall of abdomen.

Though there is no doubt that the maintenance of an effective collateral circulation postpones the evil day, the occurrence of ascites must not be regarded as absolute evidence of its failure. The appearance of peritoneal dropsy is rather the indication of the climax of the general deterioration, and is frequently preceded by or associated with other symptoms, such as dyspnœa, palpitation, anasarca, albuminuria, more or less jaundice and gastro-intestinal disturbance, indications not only of the hepatic inadequacy, but of the participation of the heart and kidneys.

The clinical pictures of declared cirrhosis vary much as regards some important features, the principal three being the size of the liver, ascites, and jaundice. The variations in weight and bulk of cirrhotic livers are great, so too are the variations as to the occurrence and amount of jaundice and ascites. Though the larger the liver the more marked the jaundice, and the smaller the liver the more marked the ascites, is a rule which holds fairly true, yet the liver

may be large, the ascites great, and the jaundice slight. There is not sufficient ground for making a hard and fast boundary between the livers of atrophic and hypertrophic cirrhosis; they are the same disease, produced by the same etiological factor acting on organisms with different resistances.

The detection of the hypertrophic liver is attended with less difficulty than the discovery of the size of an atrophic organ. It can be felt, percussed out; it is probably tender, there is marked jaundice. The size of the small cirrhotic liver is often masked by the presence of ascites, and may, from being pushed or dragged down, seem larger than it really is, and this is especially true of the intermediate grades, where both jaundice and ascites are prevalent together.

After a more or less prolonged period of symptoms characterised by gastro-intestinal irritation and congestion, as evidenced by vomiting, epistaxis, hæmatemesis, piles, and so on, the patient's attention is called to his increasing girth. For some time past he has been getting progressively debilitated, complaining of weakness, shortness of breath, and loss of flesh. The hepatic facies has become more and more marked. If inspected in the erect posture it will be noticed that the abdominal enlargement forms a downward bulging if due to ascites, with diminished bulk of liver; while, if the patient be jaundiced and the liver enlarged, there will be an obvious bulging of the upper part of the abdomen chiefly to the right.

The amount of fluid in the peritoneal sac may vary from a few to many pints. In character it is usually a thin, easy-flowing, pale yellow fluid, but may be of darker hue from staining with bile or blood. The specific gravity is variable, but mostly ranges from 1010-1015. The reaction is alkaline. Soft, fleecy-looking fibrinous coagula often form in it after standing.

The presence of this fluid in the sac causes discomfort, and the early stages of its accumulation are often associated with marked tympanites from gaseous distension of the paretic intestine. As the amount increases it interferes with the action of the diaphragm, causing palpitation, dyspnœa, and cough, and is frequently associated with increased intestinal trouble, and with the appearance of anasarca. The collection of a notable amount of fluid is usually attended by marked ingravescence of the symptoms and progressive debility. The patient becomes thinner and weaker, his features look more pinched, and his face more hepatic than ever. The cardiac action keeps on getting feebler, and the loss of appetite and distaste for food increase. The terminal stages of these cases vary. Some die

of progressive asthenia, others after hæmorrhage; some from intercurrent disorders; some from the effect of associated disease such as cardiac, renal, or tuberculous; but most from hepatic inadequacy, with cerebral symptoms, coma, delirium.

In cases of contracted liver jaundice is a less marked feature than when the liver is large; but it is much more common, especially in the later stages, than is usually credited. On the other hand, when jaundice is a prominent feature, and the liver large and bulky, the presence of ascites is a variable phenomenon. Sometimes it is conspicuous by its absence, but often enough there is bile-stained ascitic fluid, sometimes in considerable quantity, in the peritoneal sac.

During the preliminary stages the varieties of cirrhosis present very similar symptoms, but when the indurative process has reached a certain stage differences become apparent in the clinical picture. The enlarged or hypertrophic variety can be made out easily, indeed, the bulging in the right hypochondriac and epigastric regions is often sufficiently obvious on inspection. Though increased in size the organ preserves its natural outlines. It is tender on pressure, and the patient often complains of pain, dull, aching, or fitfully severe. There are often slight rigors, followed by rise of temperature, 102°-104°, and the pyrexia may be of daily occurrence. Though the jaundice may be deep, the stools may still be coloured with bile; but when it has once set in it goes on increasing, either gradually or by fits and starts. These are the cases which so frequently terminate as examples of hepatic inadequacy, the end being marked by delirium, stupor, and coma.

In addition to the condition brought about by the morbid changes occurring in the liver, the clinical symptoms may be aggravated, or even masked, by associated disease of other organs. The coexistence of Bright's disease is a well-known accompaniment of cirrhosis, but the degenerated condition of the heart is not always fully appreciated. There is often not only excess of subepicardial adipose tissue, but fatty degeneration of the cardiac muscle as well, as breathlessness, palpitation, feeble and indistinct sounds may indicate clinically. The occurrence of albumin in the urine from the presence and pressure of ascitic fluid in the peritoneal sac has been frequently noted. This may disappear after tapping, but when it persists, and is accompanied by the presence of casts, it is an indication of organic changes in the kidney.

Many cirrhotics are affected with pulmonary phthisis, with its usual accompaniments of cough, expectoration, and other symptoms, with intestinal tuberculosis and tuberculous peritonitis.

Though the clinical picture has been taken from cases of cirrhosis where alcohol has been the exciting etiological factor, the description holds good in the main for other fibrotic conditions, whether the new formation has taken place around the individual lobules, has included many, or is irregular or even intercellular in distribution. According to the writer's observations, it would seem that cirrhotic livers are, quite if not more, frequently bulky than the reverse; that jaundice, even when the liver is decreased in size and weight, is much more common than is usually taught; that ascites is the frequent companion of cirrhosis with jaundice; that cirrhotic livers may range from 30 to 130 ozs. in weight; and that enlargement of the spleen is a factor common to all classes of cirrhosis, and occurs in about 75 per cent of cases. And it seems an easy inference that cirrhosis is a general and systemic degeneration, as well as one expressing itself by symptoms of hepatic inadequacy.

Extreme enlargement of the liver from the prolonged use of alcohol, combined with hypnotics, may suggest hypertrophic cirrhosis. I can recall three such cases where the prominent features were jaundice, great enlargement of the liver, and hepatic tenderness. These livers weighed over 7 lbs. apiece, were smooth and yellow, and histologically revealed fatty degeneration, but not any evidence of cirrhosis.

The hypertrophic biliary cirrhosis of Hanot is more frequently talked about than seen, and different writers seem to hold different views as to the definition of the disease. According to the original monograph there is a variety of hypertrophic cirrhosis, which is characterised by extra- and intra-lobular fibrosis, without any tendency to contraction, and exhibiting an abnormal development and catarrh of the biliary canaliculi. It is a disease marked by its slow progress, by chronic jaundice, by enlargement of the liver, by the absence of ascites, and of evidence of external collateral circulation, and often ending in icterus gravis. Now there is nothing in this *resumé* which differs materially from the ordinary story of the large cirrhotic liver due to alcohol; and as nearly all the cases quoted by Hanot were obviously alcoholics, and quite half of them had ascites, it seems difficult to believe that there is any necessity for protracting the existence of this somewhat mythical disorder.

But it would be illogical to deny that other toxins than alcohol might produce cirrhosis, and it is perfectly conceivable that their path of action may be an ascending one from the intestine, as well as a descending one from the portal, and even by the hepatic artery

blood. The researches of Adami on the cirrhosis of cattle, the occurrence of infectious and epidemic jaundice, the occasional appearance of a variety of hypertrophic liver associated with a peculiar bronzing of the skin and often with diabetes, and some recorded instances of irregular types, all point in this direction.

The *syphilitic* virus may participate in producing indurations and enlargements of the liver. In the acquired form the general symptoms resemble in the main those of alcoholic cirrhosis, and it may be that both conditions coexist. The liver itself is usually enlarged, irregular, and tender. The unevenness may be due to gummatous deposit, or to the contraction of inflammatory fibrosis, while the tenderness is often connected with perihepatitis, and may then be associated with pyrexia. A diagnosis of syphilitic cirrhosis may be made, if, in addition to the foregoing, there be a history of syphilis, or evidence of the disease in the skin, mucosas, etc.

In cases of *congenital syphilitic cirrhosis* the infants are mostly brought by their parents for weakness, emaciation, and vomiting. There is usually a syphilitic rash on the trunk and limbs. They are sallow, withered-looking objects, usually suffering from bronchitis and running from the nose. Jaundice may or not be present, but occurs later. There may be ascites and pyrexia. Unless there be jaundice or an obvious enlargement, say in the epigastric region, from the presence of a gumma, the diagnosis is usually made post-mortem. Often, indeed, this intercellular hepatitis is not only latent, but masked by symptoms of disease of other organs, such as kidney, intestines, or lungs.

Malarial cirrhosis is seen to present most of the features of ordinary cirrhosis. The liver is variable in size, and may present, in addition to the ordinary histological appearance, pigmentation and lardaceous degeneration. In these cases there is always a definite history of malaria; the malarial cachexia is usually present, and there is frequently a history of high living and the prolonged use of alcohol.

Diagnosis.—One of the conditions most likely to be confounded with cirrhosis is chronic capsulitis of the liver and the spleen, a condition mostly associated with more or less chronic peritonitis. The chief symptom is ascites, jaundice being only an occasional complication. In these instances a diagnosis may be arrived at, after tapping, from the shape of the liver, which is found to be round or oval.

Chronic simple peritonitis, associated with subperitoneal fibrosis, produces ascites and other symptoms suggestive of cirrhosis; in

these cases the intestines are pulled up *en masse* owing to the shrinkage of the subperitoneal tissue.

Thrombosis or obliteration of the portal vein will give rise to symptoms which are common to cirrhosis. Cases of gradual and sudden obstruction are recorded. In the former the duration has been protracted, and the evidence of collateral circulation well marked. In the latter, which may happen as a complication of cirrhosis, there has been rapid and extreme ascites. But whether the onset be gradual or the reverse, the diagnosis must be chiefly speculative, unless the rapid recurrence after tapping be regarded as a guide.

Malignant growths in the liver produce in most cases marked enlargement and irregularity of outline. Jaundice is present in quite half the number; ascites is less common. The main points of distinction are that the emaciation and debility are much more rapid than in cases of hepatitis, while, if the deposit be secondary, much assistance is derived from the knowledge of cancer existing elsewhere. Still there are periods when it is difficult to be certain whether we have to deal with hypertrophic cirrhosis or a cancerous condition, more especially when the latter supervenes in an organ already cirrhotic, as it sometimes, though very occasionally, does.

Cases of large cirrhotic liver, with jaundice and pyrexia of a pyogenic type, have been mistaken for abscess, and this eventuality is more likely to occur in malarial cases, and where the patient has suffered from dysentery.

The **prognosis** of cirrhosis in all cases is certainly bad, more especially so when either ascites or deep jaundice, or both, are predominating features. Given the fact that cirrhosis exists, it is mostly a question of time, and the duration depends very much on how long the compensatory circulation remains effective. That it may remain in working order for a considerable period, even years, is certain, but when the hepatic derangement is complicated with associated conditions, these latter are of paramount importance in estimating the prognosis of any given case. When there is evidence of Bright's disease, of heart mischief, of the presence of tubercle in the lung or elsewhere, it is obvious that the outlook becomes graver. As defects of the heart and kidneys are of themselves capable of producing dropsy, it is not wonderful that when either or both co-operate with a damaged liver that ascites and anasarca rapidly supervene. When tapping has to be resorted to, it is usually a question of a few months only.

But though the vast majority of cases have a short duration after

the onset of ascites, some do comparatively well after repeated and early tapping. Some of these no doubt are syphilitic, or cases in which syphilis is a predominating factor; in others, the disease may remain stationary, probably owing to cessation from the use of alcohol.

As it is usually impossible to detect the early stages of cirrhosis, and the diagnosis of this condition is speculative, or merely a matter of suspicion, the **treatment** must have reference to the symptoms complained of, which usually are those of alcoholism, and a mixture of nervous and gastro-intestinal derangement. Should, however, there be symptomatic evidence pointing to or strongly suggestive of cirrhosis, as indicated by some enlargement of the liver and hepatic tenderness, the presence of piles, epistaxis, a "liverish" appearance, and supported by a history of alcohol, the hope of preventing further induration and arresting the progress of the fibrosis should be held out, and the cessation from the use of alcohol insisted on. A bland and easily assimilable dietary is to be ordained; red meats, rich or highly-spiced dishes and condiments, are to be prohibited. The most suitable fare appears to be milk, eggs, fish, and fresh fruit and vegetables. Milk, however, even when diluted with soda or Vichy water, etc. is often borne badly by adults, and then may be replaced by whey, thin soups, or some of the numerous predigested foods.

Mineral waters, especially those of a laxative nature, are often agreeable to the patient, and useful also as remedial agents. The natural springs which are in repute for the relief of diseases of the liver are too numerous to be listed in full, but the names of the following may be mentioned—Matlock, Harrogate, Leamington, Carlsbad, Vichy, Contrexéville, Neuenahr, Marienbad.

The bowels should be kept easily and regularly open by means of mild aperients, by the use of fresh fruits and vegetables, aided by exercise, with the occasional assistance of enemata. Strong aperients should be avoided, not only on account of their weakening effect, but also on the ground that, as the absorptive function of the alimentary system is deteriorated, greater time is required for intestinal digestion. *Per contra* in many cases the reverse condition has to be dealt with, and the use of astringents may be compulsory.

Though no form of treatment avails to remove fibrous tissue, much less to regenerate a cirrhotic organ, the use of iodides has in some instances been followed by remarkable results. In all cases where there is a syphilitic history, and indeed even when none is obtainable, iodide, combined with mercury, should be exhibited, but

too much must not be expected from their use. In quite small doses mercury acts as a tonic, while the combination of iodide and quinine is at times as surprising as at others it is disappointing.

The best general tonic, especially when the heart is involved, is digitalis.

Vomiting and hæmatemesis should be treated by complete rest of the body and the stomach. For both, the hypodermic injection of morphia is often useful, and while the former may often be controlled by means of hydrocyanic acid and soda, for the latter rectal alimentation should be employed until, by tentative feeding by the mouth, we are assured that the stomach can bear its ordinary diet. The supervention of coma is best met by means of saline transfusion, for, though of only temporary value, it may delay the inevitable end.

The appearance of ascites is an indication for surgical measures. Until recently strong purgatives and strenuous diuretics were much in vogue, and paracentesis was postponed as late as possible; but quite the reverse method now obtains. Far more favourable results follow early tapping, provided the disease is not too far advanced, and the associated morbid conditions not too severe, than would have been expected before the advent of aseptic surgery. Late in the disease, when the patient is exhausted or worn out, or when the heart and kidneys are also inadequate to fulfil their functions, evacuation of the fluid can only afford a temporary relief.

Very favourable results have been obtained from laparotomy and stitching the liver to the anterior abdominal wall, with the view of opening up a compensatory collateral circulation through the adhesions: results which suggest that perihepatitis ought to be a favourable combination.

HYDATID OF THE LIVER

Symptoms.—In the early stages of their development hydatid cysts do not give rise to any symptoms. As they increase in bulk their presence may be attended by feelings of discomfort, but it is not until definite evidence of pressure on surrounding parts or organs, by the discovery that there is undue prominence of the epigastric or hypochondriac regions, or by the more or less sudden occurrence of one of those accidents to which these cysts are liable or give rise, that attention is attracted towards the liver. In a large proportion of patients the presence of hydatid cysts

does not cause any material deterioration of health. There is rarely any emaciation, debility, pyrexia, pain, or jaundice, but in certain instances some loss of flesh, pain, and jaundice have been noticed. Practically, however, it is either some symptom indicative of pressure on adjacent structures or increasing girth which eventually directs attention to the liver. Thus, pressure on the base of a lung may give rise to cough and dyspnœa, on the heart to breathlessness and palpitation, on the cava or portal vein to anasarca or ascites, on the stomach or intestine to gastro-intestinal symptoms.

On examination the liver is found to be enlarged, and, when the cyst is situated towards the front, a definite bulging or rounded boss may be detected in the upper part of the abdomen on inspection and palpation. This swelling, more or less hemispherical in shape, may project from the upper or lower surface. It moves with the liver. It is dull on percussion. Sometimes it is hard and rigid, feeling like a solid tumour, at others it imparts a distinct sense of fluctuation, and it is said that occasionally a characteristic vibration (hydatid fremitus) may be obtained by percussion. The swelling feels quite smooth to the touch, its outline is regular, and it is not tender on handling. Should it be situated on the posterior half of the liver, the determination of its size and characters is more than difficult. There may be bulging of the lower part of the chest; the liver is enlarged and pushed somewhat downward and forward; there may be impaired resonance or even dulness, with diminished respiratory sounds at the base of the lung. Later the exudation of fluid (passive or inflammatory), as in other cases of subphrenic swelling, ensues, and the nature of the case is probably revealed by paracentesis.

Though there may be only one cyst, there are frequently two or more. When more than one the liver outline may be obviously uneven. In size they vary extremely, and in situation are more common in the right lobe.

Hydatid cysts are liable to certain complications and accidents which are attended with dangerous, often fatal sequelæ. They may be ruptured from traumatism. Should the cyst burst, as it often does, into the peritoneal sac, it may excite acute peritonitis. This sequel is, however, not invariable, but the consequence may be that the peritoneal sac is infected with scolices, and later becomes the site of one or more cysts, the mother cyst drying up. They may suppurate, the remains being found as a shrunken, tough capsule containing putty-like or calcified debris. Should, however, the pus make its way outwards it may excite pneumonitis, pleuritis,

peritonitis, according to the direction it takes and the original location of the cyst, or even pyæmia. They may ulcerate through into a large bile duct, in which case they cause jaundice accompanied by colic, and they have been known to burst into the pericardial sac, into the portal vein, and into the inferior cava, with rapidly fatal results. They have made their way into the stomach and the intestine, the contents of the cyst having been vomited up or voided per anum. They have been ruptured during labour, and given rise to peritonitis. As a secondary consequence of rupture, and consequent sowing of the peritoneal sac with daughter cysts, instances are recorded where they have caused dystocia from pressure on the uterus and vagina, and retention of urine from pressure on the bladder.

An erythema or urticaria sometimes follows the absorption of fluid which has escaped from a ruptured or punctured sac.

The **diagnosis** of hydatid of the liver may be comparatively easy, but is often attended with considerable difficulty. It may be easy if the patient be in good health, if there be no emaciation, no loss of colour; if the patient be fairly young; if there be no history of syphilis; if the swelling be painless, of gradual growth, of long duration, if it be cystic in character and situated towards the front of the organ. When situated posteriorly the diagnosis becomes more difficult. In these deep-lying parts the associated symptoms and conditions are increased size of the liver, which is pushed downwards and forwards, and discomfort arising therefrom; dyspnœa and cough from pressure on the base of a lung; but until inflammatory sequelæ arise in the thorax the patient remains constitutionally well, and there is no pyrexia. There is usually some bulging of the thoracic wall, and when inflammation of the lung and pleura occurs the diagnosis is more apt to be pleurisy or pneumonia than hydatid.

Should the cyst project downwards it may simulate a distended gall-bladder, but the latter condition is usually preceded by biliary colic and is more mobile. Enormous cysts taking a downward direction have resembled ascites and ovarian cysts.

A diagnosis of ruptured hydatid becomes possible if there be a history of antecedent painless swelling, the sudden subsidence of which has been followed by symptoms indicative of the direction of the rupture.

Suppuration of a cyst gives rise to the symptoms and signs of pyogenic inflammation, which may be associated with special phenomena, *e.g.* those of pleurisy, pneumonia, peritonitis. It may easily

be mistaken for abscess of the liver; but the previous history of the swelling and the absence of ulceration of stomach or of intestine, or of malaria, may throw light on the obscurity.

The conditions most likely to be mistaken for hydatids are cancer and syphilis. In cancer there are almost invariably marked and progressive constitutional symptoms, and frequently jaundice and pain. The liver is hard, tender, irregular of surface, and does not impart any sense of fluctuation except in those rare instances of cystic and of fast-growing cancers.

In syphilis a large gummatous tumour has suggested the presence of a hydatid cyst, but syphilomata are much less regular in shape, and they are often tender. There may be some pyrexia, and there is the syphilitic history, with the probability of well-marked symptoms elsewhere.

As part of the diagnosis tapping the cyst is sometimes resorted to. The character of the fluid in these cysts is always significant, and when accompanied by the presence of membrane, hooklets, or scolices, the diagnosis becomes certain.

The **prognosis** will depend mainly on the complications and situation. When the cysts have not excited changes in neighbouring viscera, or are limited to the liver, or they are not suppurating, the prognosis should be favourable as to the event of an operation. When suppuration has occurred the outlook is less favourable. Position has much to do with the event, for those in front are more easily got at than those situated behind, and in addition the latter have great tendency to involve the lung.

Sometimes they undergo spontaneous cure and wither up, a fibrous or fibro-calcareous mass remaining to mark the site, and in this connection it may be mentioned that living and dead cysts are sometimes found after death, their existence having been unsuspected during life.

Treatment.—The treatment is purely surgical and, notwithstanding the fact that some cysts undergo what may be termed a spontaneous cure or involution, their removal should always be advocated.

Though puncturing a cyst is often practised, this procedure is not advisable; on some occasions it has been followed by fatal results. Aspiration is less dangerous. The most successful treatment is by incision, and allowing the sac to granulate up from the bottom.

GALL-STONES

Symptoms.—It is often too easily assumed that biliary calculi are formed only in the gall-bladder, probably from the fact that they are found there. This view, however, overlooks two important points: (1), that it is not infrequent to find biliary sand, often in considerable quantity, the grains occasionally being of quite large size, in the intra-hepatic canals, and (2), that such sand granules usually form the nucleus of the common facetted calculi. Hence it becomes probable that a large proportion of calculi are initiated within the liver, and that in the bladder they assume their well-known characters from the superposition of constituents derived from the bile itself, and from the secretion of the vesical mucosa.[1]

The general symptoms of their presence are of all degrees of severity. There are evidences of gastric irritation, anorexia, nausea, vomiting, a sense of discomfort or actual pain, radiating upwards and backwards, and attended with some enlargement and tenderness of the liver, with chills and even actual rigors. In such attacks the hue of the patient is often "liverish" or icteroid. The motions are often pale from deficiency of bile, due to catarrh of the ducts. Some patients are subject to frequent recurrences of these attacks.

The process of gall-stone formation is, however, in most cases unattended by pain or other symptom to call attention to their existence. It is quite a common thing to find them post-mortem in cases where they have not been suspected during life, and the bladder and cystic duct may be choked with them.

On the other hand, should a calculus become engaged in the cystic duct it may give rise to symptoms, and if it block the duct the following consequences may arise:—The gall-bladder may shrink up; it may become dropsical from the mucosa continuing to secrete; suppuration may ensue, or the gall-stone may ulcerate through the wall. Should it travel onward, its passage to the intestine may be attended by "biliary colic." If it fail to reach the gut it may get impacted in the common bile-duct or in the ampulla of Vater, and thus implicate the pancreas. The damming up of the biliary and pancreatic juices are, however, not the only consequences of impaction below the cystic duct, there are always the possibilities of

[1] These remarks are not intended to refer to calculi composed entirely of cholesterin, which, as far as the writer's experience goes, are rare and found only in the gall-bladder or its duct.

suppuration, ulceration, and, when the stone is of large size, of intestinal obstruction.

Though a gall-stone may successfully migrate from the bladder to the intestine without causing any symptoms, its transit is usually accompanied by a train of symptoms known as "biliary colic." The principal features of an attack are sudden and violent pain, radiating from the hepatic region laterally, backwards and especially upwards towards the scapula. The patient rolls about in agony, sweats profusely, and vomits frequently. Shivering or actual rigors, with pyrexia, feeble action of the heart, are constant accompaniments of the attack, which may last from a few hours to several days. During the paroxysm the liver is tender and the abdominal muscles rigid and contracted.

If the calculus pass on to the common bile duct jaundice results. This symptom, which is present in the majority of cases of biliary colic, is variable in intensity and in the time of its appearance. It may be evident in the course of a few hours, or be delayed till the second day. When the gall-stone reaches the gut the paroxysm ceases, but a soreness and dull aching in the right hypochondrium and tenderness of the liver may remain for a time. The jaundice usually subsides with comparative rapidity, but sometimes lasts for weeks, and occasionally has been permanent. Though there may be only one, subsequent attacks are not infrequent in the same patient. In some cases the symptoms are so severe that collapse and syncope ensue, and even fatal cases are reported.

When the calculus is arrested in some part of the cystic duct the frequent sequel is shrinkage of the gall-bladder, which gradually becomes a practically obliterated fibrous or even calcified bag with or without other calculi.

An alternative event is that the mucosa may go on secreting, and in this way is produced a globular or pyriform pendulous tumour depending from the under surface of the liver. Sometimes the presence of a gall-stone is associated with suppuration of the bladder, or it may ulcerate through the wall and be followed by peritonitis.

If the calculus become impacted in the common bile duct jaundice becomes a permanent feature, and the discoloration is more or less marked, or subject to variations of intensity, according as the stone is tightly jammed, or owing to its shape allows bile to flow past, or is able to slip up and down.

An important effect of a calculus located in this position is to cause dilatation of the bladder and of the ducts above the obstruc-

tion. The walls of the ducts may become thickened, and if suppuration occur their mucosa may be converted into a pyogenic surface, and so in its turn give rise to the formation of local abscesses or a general septicæmia. Sometimes the calculus ulcerates through and may find its way into the gut, but otherwise it sets up peritonitis.

The general symptoms produced by a gall-stone impacted in the common duct are pain, jaundice, rigors, pyrexia, sweating, with or without a history of antecedent colic. These symptoms become all the more important and dangerous when they are connected with or originate from a microbic infection ascending from the intestine. Microbic infection is more prone to ensue when the impaction is not complete, or is low down in the common duct or in the ampulla, owing to the pathological changes which necessarily ensue from the irritation produced by the calculus.

Diagnosis.—The diagnosis of biliary colic is often easy, and when jaundice supervenes upon the prominent symptoms it becomes practically certain. In doubtful cases the stools may be searched by diluting them with water or weak carbolic solution and straining through a fine wire sieve or coarse canvas, but the failure to find a calculus does not necessarily negative the diagnosis. Much assistance is derived from a history of previous attacks. The chief conditions which may give rise to doubt, especially in the early stage, are lead colic, right renal colic, appendicitis, and even non-calculous infectious cholecystitis. Infectious cholecystitis without the presence of gall-stones is very rare ; the symptoms are, however, much the same as those of biliary colic, with those of peritonitis superadded. In lead colic the pain is not associated with tenderness, there is no pyrexia, there is probably a blue line on the gums, and a history of constipation and of lead contamination. In renal colic the pain is lower down and shoots towards the groin and testicle, there is often great tenderness along the course of the ureter, there is blood in the urine, and there is no jaundice. In appendicitis there is a swelling in the right iliac region, while the pain and tenderness are mostly in and near the vicinity.

Some neurotic women are prone to attacks of abdominal pain accompanied by vomiting. Though the symptoms may appear to be urgent and suggestive of biliary colic, more especially as gall-stones are much more frequent in females than in males, there will be no collapse, no pyrexia, no jaundice, and *per contra* there will be usually a history of hysterical attacks and the presence of hysterical symptoms, such as globus and polyuria.

The number of attacks to which a patient is liable is extremely variable; there may be only one, but often they recur from time to time. The possibility of a future attack may be inferred from the shape of calculi recovered from the stools, for if facetted there are probably more to come, but if round or oval a more favourable view may be taken.

Treatment.—The paroxysms of extreme pain should be treated with hypodermics of morphia, or by the inhalation of chloroform, or by both combined. For lesser degrees of pain opium or belladonna suppositories, hot baths and fomentations are useful. Copious draughts of warm or hot water, to which bicarbonate of soda may be added, are thought to assist in relieving the symptoms. For the vomiting hydrocyanic acid and soda is the best remedy, but cocaine hydrochloride, ammonium bromide, and oxalate of cerium have been administered with advantage. The jaundice is sometimes attended with a distressing pruritus, for the relief of which warm baths, washing with some alkaline soap, bathing the surface with very weak carbolic lotion, or the injection of pilocarpin may be tried.

Between the attacks attention should be paid to the diet. That best suited to these patients is composed chiefly of nitrogenous substances, the starchy and saccharine being cut down to the minimum consistent with physiological principles, the object in view being to favour the formation of bile salts, and prevent as far as possible the deposition of cholesterin.

The use of alkaline mineral waters is advisable, and they may be drunk freely between meals. Moderate exercise out of doors should be enjoined. The bowels should be kept open by the aid of laxatives and mild aperients. Some of these cases seem to be relieved by the occasional employment of blue pill and haustus sennae co.; others by the use of salicylates or of sodium salts.

No success need be anticipated from the use of solutions intended to act as solvents. Massage should not be tried.

Under certain circumstances it is proper to suggest an operation for the removal of calculi. These are when the gall-bladder is distended, when jaundice is persistent, when attacks of biliary colic are of frequent occurrence, when there are indications of suppuration or of ulceration.

CHOLECYSTITIS

Symptoms.—Inflammation of the gall-bladder is almost always associated with the presence of gall-stones; occasionally, however,

it becomes inflamed from the ascent of microbes from the gut. The inflammation is either catarrhal or suppurative. In the catarrhal form the gall-bladder becomes more or less distended with viscid mucus, and sometimes attains an extraordinary size. A distended gall-bladder forms a hemispherical or pyriform tumour attached to the under surface of the liver, moves up and down with respiration, may be pendulous, and is easily moved from side to side. Its contour may be smooth or somewhat irregular; on palpation it may impart the impression of being cystic, or it may feel stony hard. It is usually very tender and often painful. The symptoms vary according as to whether gall-stones are present or not, and whether the inflammation is infective and suppurative or not. No symptoms may arise from mere distension of the gall-bladder; but the milder cases of cholecystitis are associated with discomfort or tenderness in the liver and gall-bladder, with chills, slight gastric irritation, and an icteroid discoloration of the skin and conjunctiva. When pus forms there are usually distinct rigors with hectic pyrexia, considerable pain in the liver, and tenderness over the gall-bladder. Unless the bile ducts are simultaneously involved jaundice does not occur.

A positive **diagnosis** of this particular condition is more or less speculative, for, on the one hand, it may be mistaken for gall-stone colic plus inflammation, and on the other for peritonitis and intestinal obstruction. The main symptoms to rely on are pain, extreme tenderness in the region of the gall-bladder, and the presence of a tumour in this situation, vomiting, pyrexia, with distension of the abdomen. There may be no jaundice. As cholecystitis may be the sequel of or be accompanied by pancreatitis, careful examination should be made for the presence of fat in the stools and of sugar in the urine. Though these bodies are usually absent under these circumstances, their presence when they do occur is of great diagnostic importance (see pp. 370, 377).

The **treatment** consists in opening and draining the gall-bladder. Sometimes, however, the symptoms subside without recourse to this radical measure, and their disappearance is thought to have been hastened by the use of purgatives, of anodynes, and local applications.

ANGIOCHOLITIS

The bile channels from their intercellular origin in the lobules to the termination of the common duct may be the seat, in part or

throughout, of inflammation set up by extension from the bowel, or, as Dr. W. Hunter suggests, originating in the smaller passages from the irritant character of substances derived from the digestive tract, that may be excreted in the bile. The inflammation may be of a simple catarrhal, or of an infective character, the latter accompanying many infective disorders, as well as septic processes taking place in the liver and gall-stones.

In the mildest degree the catarrh gives rise to the condition known as "biliousness," and, as already described, "toxæmic jaundice" is attributed to obstruction in the smallest ducts caused by catarrh (p. 419).

Suppurative angiocholitis is a grave malady marked by high fever, rigors, and jaundice, which may occur in periodic attacks according to the nature of the cause. Post-mortem minute abscesses may be seen disseminated through the liver, sometimes involving branches of the portal vein and giving rise to pylephlebitis (see p. 551).

For the simple catarrh a dose of calomel or blue pill, followed by a saline aperient, is usually sufficient, requiring, it may be, repetition in a couple of days; an alkali or acid and bitter mixture may be given, or an effervescing draught every few hours if nausea and sickness predominate. A light fluid diet should be ordered. For the suppurative condition little can be done except so far as the cause is removable.

CANCER OF THE GALL-BLADDER AND DUCTS

The **symptoms** due to cancer arising in the gall-bladder are at first indefinite, and their sooner or later development probably depends on the trend and direction of the new growth. After a period varying from weeks to months, during which the patient becomes weak and loses flesh, complains of pain between and in the shoulders and down the back, nausea and occasional vomiting, he becomes more or less rapidly jaundiced. On examination it is found that a rounded mass, not separable from the liver, can be detected in the right hypochondriac region. The patient goes rapidly downhill, and dies from hepatic inadequacy, asthenia, or septicæmia, the end being hastened by one of the numerous complications of cancer of this structure, such as extension to the peritoneum, pressure on the portal fissure, ulceration, and even sloughing of the malignant mass. Though the foregoing—viz. emaciation, weakness, pain, local and radiating to the scapular region, presence of a rounded

tumour connected with the liver—may be regarded as the cardinal symptoms, some may be absent or but little marked. There may be no jaundice, and but little pain or emaciation.

Sometimes there is pyrexia, and this may be unconnected either with gall-stone irritation or inflammatory complications. Sometimes gastric symptoms, nausea and vomiting, are features throughout.

During life it is more than difficult to distinguish between cancer beginning in the bile ducts and in the gall-bladder, for the general and local symptoms are much alike. The early occurrence of jaundice is the main distinguishing feature.

Occasionally no tumour connected with the liver can be detected, but as a rule there is obvious enlargement of the gall-bladder, and in most cases the presence of gall-stones has to be reckoned with. When the growth starts in the ampulla the pancreas is almost necessarily implicated, and hence symptoms indicative of diabetes may supervene.

The **diagnosis** of malignant disease of the gall-bladder and bile ducts has to be made mainly from inflammatory and irritative conditions arising out of the presence of gall-stones and from malignant disease of the head of the pancreas.

Many examples of cancer of the gall-bladder are diagnosed as primary cancer of the liver, and this naturally occurs when, after the liver is invaded, the growth rapidly extends and forms a prominent tumour. Before the liver is invaded the symptoms may have been so slight that the onset of the disease is made coincident with that of the hepatic derangement.

The age of the patient is greater in malignant than in inflammatory conditions, for cancer of the gall-bladder is rare before forty.

The diagnosis of cancer of the gall-bladder and bile ducts from that of the head of the pancreas must depend in great measure on the site of the tumour, for most of the symptoms due to malignant disease of these parts are much alike. There is pain, emaciation, debility, and jaundice, probably some pyrexia and gastric symptoms. In cancer of the gall-bladder the tumour is attached to the liver, is comparatively superficial, and moves with respiration. In disease of the pancreas, if a tumour can be felt at all, it is deep-seated and fixed. In a good many cases of disease of the pancreas some information may be derived from an examination of the stools and urine, for the presence of fat in the motions and of sugar in the urine has been detected.

The conditions arising from gall-stones are the result of their

presence mainly in the cystic or common ducts, of their accumulation in the bladder and the inflammatory sequelæ thereof. Here biliary colic, ague-like attacks, the urgency of the gastric symptoms, the age of the patient, and the absence of previous emaciation and debility, afford the main distinguishing criteria.

The **prognosis** is always the gravest if the existence of cancer be certain. In doubtful cases an exploratory laparotomy is advisable, as growths have been removed from the duct with some success.

The **treatment** must be directed towards the alleviation of symptoms. The line to be adopted is the same as that indicated in new growths of the liver.

R. G. Hebb.

DISEASES OF THE PERITONEUM

The Peritoneum is not only the seat of disease with which it is primarily attacked, but from its extensive relations is very liable to become secondarily involved, either generally or only over limited regions. The affections of this structure present in their clinical manifestations the gravest features, and are the cause of death in not a few disorders of the abdominal organs.

GENERAL ANATOMY OF THE PERITONEUM

The peritoneum is a serous membrane lining the abdomen and covering more or less completely the contained organs, for some of which it forms suspensory ligaments and mesenteries. It is so disposed over the viscera as to form two sacs—the greater is that of the general abdominal cavity, the lesser is behind the stomach, the two being connected by a narrow neck called the foramen of Winslow.

The edges of this foramen are probably always in apposition, and even when stretched it will not admit more than two fingers. It has in front of it the hepatic artery, the portal vein, and common bile duct, behind the vena cava, below the duodenum, and above the caudate lobe of the liver. As the entrance to this lesser sac of the peritoneum is so small it may be occluded by inflammatory processes and then localised effusion may take place therein. The exact boundaries of the lesser sac are as follow :—

In front, at the level of the foramen of Winslow, is the gastro-hepatic omentum, above the spigelian lobe of the liver, below the stomach, from the posterior surface of which the peritoneum passes down and helps in the formation of the great omentum. Above, the limit of the lesser sac is the line at which the posterior surface of the liver comes into contact with the spine ; from there downwards its posterior limits are the spine with the structures on it, the left crus of the diaphragm, the pancreas, and the cœliac axis ; leaving the spine it becomes the upper layer of the transverse mesocolon, and, having invested the upper surface of the transverse colon, passes into the great omentum, where it becomes continuous with the layer we have already seen descending from the posterior surface of the stomach. The limits above mentioned are those in or near to the middle line, but in its extent to the left, as far as the spleen, it is bounded in front by the posterior wall of the stomach and the gastro-splenic omentum, behind by the left kidney

and suprarenal capsule, and between the kidney and the spleen it forms the anterior layer of the lieno-renal ligament.

The relation of the viscera to the peritoneum is of importance clinically. The *stomach*, except immediately behind the cardiac orifice, where it is in direct contact with the diaphragm, is completely enveloped by this membrane, which passes from the smaller curvature to the under surface of the liver, forming the gastro-hepatic omentum, and from the greater curvature to form the anterior part of the great omentum, the posterior surface of the stomach thus constituting a part of the wall of the lesser peritoneal sac. The reflection of peritoneum from the cardia to the diaphragm is known as the gastro-phrenic ligament, and that which passes from the fundus to the spleen, in which run the gastric branches of the splenic vessels, is termed the gastro-splenic omentum. In contrast to this arrangement the *duodenum* is only very partially covered, the posterior surface throughout being more or less closely connected with the post-peritoneal tissue in the neighbourhood of the head of the pancreas, kidneys, and large vessels, except quite close to the pylorus, where it is completely surrounded by peritoneum; the anterior surface only of the rest of the duodenum is covered by peritoneum, save where it passes into the jejunum, and here the sides, too, are covered by it. The *jejunum*, *ileum*, *cæcum*, *appendix*, *transverse colon*, and *sigmoid flexure*, are entirely surrounded by peritoneum, and, except the cæcum, are suspended by mesenteries; the remainder of the colon is only partially covered, being more or less firmly attached posteriorly by connective tissue; the rectum, with the exception of the anterior surface of its upper portion, is devoid of peritoneal covering; in 26 per cent of cases the ascending, and in 36 per cent the descending colon has a mesocolon; in about 5 per cent the cæcum is bare posteriorly and is connected with the iliac fascia by areolar tissue. The relative fixity or mobility of the different sections of the canal in reference to the extent of peritoneal covering is of importance.

The line of attachment of the mesentery which slings the jejunum and ileum, or, in other words, the line along which the peritoneum is reflected off from the posterior wall, is about 6 inches in length, and extends from the left side of the second lumbar vertebra obliquely downwards across the fourth part of the duodenum, aorta, inferior vena cava, and right psoas muscle to the right iliac fossa; inasmuch as the bowel which the mesentery supports is about 21 feet in length, the membrane is much frilled out, like a folded fan; its average length, from the bowel to its attachment to the posterior abdominal wall, is about 8 inches, and it contains between its layers blood-vessels, lacteals, nerves, lymphatic glands, and fat.

Owing to the reflection of the serous membrane off the posterior abdominal wall on to the intestine, there occur at various places—especially at the duodeno-jejunal junction, in the region of the cæcum

and appendix, and at the sigmoid flexure—various fossæ or pouches into which portions of the bowel may slip.

The *liver* is entirely covered by peritoneum, except the posterior surface to the right of the vena cava, the transverse fissure, and the fossa of the gall-bladder. The *pancreas* is only covered on its anterior and inferior surfaces. Both *kidneys* are destitute of peritoneum behind; the right is covered in front, except at the parts on which the suprarenal body, the duodenum, and the ascending colon lie; the left is covered in front, except at the parts on which the suprarenal body, the colon, and the pancreas lie. The *suprarenal* bodies are covered anteriorly, except the upper angle of the right one which is closely adherent to the liver, and its internal inferior angle which is crossed by the duodenum, and the internal inferior angle of the left one which is crossed by the pancreas. The *ureters* lie behind the peritoneum. The *bladder* is only covered by peritoneum on its superior surface, the posterior surface above the vesiculæ seminales, and the lateral surfaces above the obliterated hypogastric arteries, so that the antero-inferior surface lies behind the pubes and abdominal wall, separated only by loose fat continuous with the pelvic and sub-peritoneal connective tissue; the lower lateral surfaces are in similar relation to the levatores ani, and the lower posterior surface is in contact with the rectum, the lower part of the vasa deferentia and the vesiculæ seminales (in the female, with the vagina and cervix uteri). The *vagina* is only covered by peritoneum on the upper end of its posterior wall. The *uterus* is covered on the whole of its posterior aspect, on the fundus, and on the anterior part of the body; the anterior aspect of the cervix is uncovered, and is in contact with the bladder. The *Fallopian tubes* are covered by peritoneum, except between the attachments of the two layers of the broad ligaments, and at its external orifice where the tubal mucous membrane becomes continuous with the peritoneum. The *ovary* is covered by germinal epithelium, which is continuous at the hilum with the peritoneum of the posterior layer of the broad ligament. The *spleen* is entirely covered by peritoneum, except at the hilum.

The *great omentum* hangs down from the lower border of the stomach and lies on the transverse colon and most of the small intestines, for it reaches nearly to the pubes, and usually extends a little farther down on the left than on the right side. It consists of four layers, with part of the cavity of the lesser sac between the anterior two and the posterior two; this is the arrangement in the new born, but in adult life the four layers usually adhere, so that the cavity of the lesser sac does not extend much below the stomach. In stout people the great omentum contains much fat.[1]

[1] The investigations of Prof. Lennander of Upsala appear to show that the parietal peritoneum, which derives its sensibility from the intercostal nerves in the subserosa, is extremely sensitive to all manipulation, and that forward traction on the mesenteries

ACUTE GENERAL PERITONITIS

Etiology.—The older writers used to describe idiopathic acute general peritonitis. There is no evidence that such a disease exists. In almost every case acute general peritonitis is due to the invasion of the peritoneal cavity by micro-organisms. The bacillus coli communis is most often found, streptococcus pyogenes is almost as common, and considerably less frequently than either of these occur staphylococci, pneumococci, gonococci and other micro-organisms, *e.g.* micrococcus lanceolatus, bacillus proteus, bacillus aërogenes capsulatus, bacillus pyocyaneus, and bacillus typhosus. Usually the infection is mixed.

The introduction of the micro-organisms into the peritoneal cavity may arise from injury with an instrument which is septic, *e.g.* a dirty dagger, but the peritoneum may be considerably injured by aseptic instruments without any peritonitis following, and of this examples are frequently seen in aseptic abdominal operations. But by far the most important mode of entry of micro-organisms is by the rupture of some cavity from which they enter the peritoneal cavity and set up what is usually called perforation peritonitis. Among twenty-one cases of this recorded by Janz, seven followed perforation of the appendix, five that of a gastric ulcer, three that of a typhoid ulcer, two that of an incarcerated hernia, and one was due to each of the following—rupture of the stomach, rupture of intestine, rupture of duodenal ulcer, and rupture of perimetritic abscess. Obviously the number of conditions which may lead to perforation peritonitis is very great, and many of them are highly exceptional, *e.g.* fatal perforation peritonitis has followed rupture of a lymphatic gland which was suppurating secondarily to an inflamed pile. The bacillus coli communis is nearly always found in the exudate in cases of perforation peritonitis, together with other micro-organisms.

The next most important form of acute peritonitis is that which follows the entry of micro-organisms from the female genital organs; indeed, in former days, owing to want of knowledge of the importance of cleanly midwifery, puerperal peritonitis was much more important than perforation peritonitis. Thus in the ten years 1875-85 Grawitz found among fatal cases of peritonitis in the Charité

or omentum, or on adhesions involving the anterior abdominal wall cause great pain. Otherwise the serosa of the viscera generally seems to be quite insensitive to touch, to thermal or to painful stimuli, and this whether the peritoneum be healthy or inflamed.

176 were puerperal and 152 were perforation peritonitis. The micro-organisms most often found in the exudate of puerperal peritonitis are streptococci, but bacillus coli communis and gonococci may take part in its causation.

Occasionally acute peritonitis follows a gonorrhœal salpingitis. In the human subject, judging from fatal cases, the peritonitis is usually and perhaps always due to a mixed infection of gonococci with other micro-organisms, most frequently streptococci. Wertheim, however, claims to have produced a pure gonococcal peritonitis in animals, but they always recovered, and it may be that the cases of gonorrhœal peritonitis which occur in women and recover are instances of a pure gonococcal infection. It is said that in the male sex peritonitis may occur with gonorrhœal epididymitis.

Experimental research goes to show that if the wall of the gut be damaged bacteria will pass through it and set up acute peritonitis, and this probably explains those cases of peritonitis which occur in association with disease of the gastro-intestinal tract without any perforation, *e.g.* perforation of the appendix is not always found when the peritonitis clearly is due to appendicitis, or, again, severe peritonitis may follow the gastro-enteritis which is secondary to swallowing an acid even if there be no perforation. The rare cases of actinomycosis of the peritoneum are probably due to the direct growth of the ray fungus through the intestinal wall. Sometimes acute peritonitis is due to rupture of some solid organ, *e.g.* spleen or liver, and it may follow diseases of the abdominal wall, *e.g.* sloughing abscess.

In all the examples of acute peritonitis just considered infection has taken place locally, but occasionally it is due to the micro-organisms being conveyed to the peritoneum by the blood. The best instance of this is pneumococcal peritonitis. Netter, in 140 observations on peritonitis, found it to be pneumococcal twice. This form is usually secondary to pneumonia, but it may be that the first lesion the pneumococci produce is acute peritonitis. It can be produced experimentally in animals. The peritonitis which has been described as occurring with erysipelas is another instance of the conveyance of the micro-organisms by the blood.

There is, however, much to be thought of besides micro-organisms when considering acute peritonitis. Experiments (*e.g.* those with methyl blue) show that in health absorption from the peritoneal cavity is very rapid, and further it is possible to inject into the peritoneal cavity sufficiently small doses of micro-organisms for no peritonitis to follow, although larger doses of the

same would induce it. In the first case no peritonitis follows, either because not enough toxines are formed or because the nutrient media of the micro-organisms are absorbed with too great rapidity, and hence they die, for if the same small doses are injected with media that are very slowly absorbed suppurative peritonitis follows. It is almost certain that in many abdominal operations, in spite of every precaution, micro-organisms enter the peritoneal cavity, but the dose of them is so small that no harm results. Then, further, there is much difference in the ease with which peritonitis is induced in different animals of the same species, and this is true also of human beings; indeed, there is a case on record in which the extravasation of masses of fæces did not set up a fatal peritonitis, but lymph was thrown out around them and they were encapsuled; also the virulence of the strain of the bacillus is of great importance. Again, the resistance of the peritoneum may be much lowered by either general depression of the vital powers, hence the frequency with which acute peritonitis occurs at the end of exhausting diseases, *e.g.* the acute peritonitis which accompanies Bright's disease, or by local lowering of its own resistance, hence the acute peritonitis which occurs towards the end of chronic tuberculous peritonitis.

Much experimental work has been done to determine whether aseptic chemical irritants can set up acute general peritonitis, in the production of which bacteria do not participate, and it appears that they can, but examples must be very rare in the human subject. There are, however, a few instances scattered through literature which have been taken to show that the escape of aseptic bile may set up acute general peritonitis; such a case is that of Laehrs, in which a carter ruptured his gall-bladder. He became jaundiced, lived three weeks, and after death there was much peritonitis and the peritoneum contained much bile-stained fluid; but in considering such a case it must be remembered that the bacillus coli communis is very commonly present in the gall-bladder. On the other hand, Fraenkel claims to have produced a bacteria-free peritonitis in rabbits by the injection of sterile bile. Not infrequently cystic adenomata of the ovary rupture into the peritoneal cavity; this by no means always leads to acute general peritonitis, but if not the peritoneum becomes infected by the new growth, which, flourishing in the peritoneal cavity, leads to chronic ascites. Some cases of extra-uterine pregnancy are instances of considerable irritation of the peritoneum without the production of acute general peritonitis. But in connection with

the whole subject of bacteria-free peritonitis, it must be borne in mind that although the irritant, *e.g.* the contents of an ovarian cyst, may be sterile, it by no means follows that the peritonitis will be bacteria-free, for the irritant may so hinder the resisting power of the peritoneum that bacteria pass to it from the blood. Some observers having found after death from peritonitis following damage to the bowels (*e.g.* strangulation) that the effused fluid is occasionally sterile, have concluded that toxines may pass through the damaged wall and set up general fatal peritonitis. If such cases really exist, they would come under the head of chemical peritonitis. Mechanical injury alone, apart from the introduction of micro-organisms, is not a cause of general peritonitis; any injury so serious that it might be supposed to set up acute general peritonitis would itself kill the patient.

Morbid anatomy. — Occasionally micro-organisms in the peritoneal cavity kill before they produce any changes in the peritoneum visible to the naked eye. This is because they rapidly form virulent toxines, which are very speedily taken up by such an excellent absorbing surface as the peritoneum. Mr. H. E. Durham suggests that in many cases of supposed fatal shock after abdominal operations death is really due to this cause.

The first visible evidence of acute general peritonitis is that the shiny surface of the peritoneum becomes dull and the peritoneal vessels are dilated, so that the peritoneum looks redder than usual, and vessels not visible in health form a tree-like network. As adjacent coils of intestine exercise pressure on each other where they touch, the redness is best seen in bands along the intestine where it is not in immediate contact with an adjacent piece, and these red bands are called "suction bands." The dulness of the surface is due to an exudation of lymph, as may be demonstrated by its sticky feel and by scraping the surface with a knife. This lymph is, like the red bands, best seen where pressure is least. The intestines are usually much distended with gas owing to paralysis of their muscular coat. If the patient lives long enough the lymph will become visible to the naked eye as a whitish opaque exudate, which is often extensive enough to cover the spleen and liver, glueing together the coils of the intestine and fixing them to one another and also to adjacent viscera; but these adhesions can be torn down by slight traction. By this time, and often earlier, tinted fluid is effused; it usually gravitates to the back of the abdomen and into the pelvis, but it may be held between coils of intestine by adhesions. At this period of

the process we find the walls of the intestine implicated; they are sodden, swollen, soft, and easily tear, and the peritoneal coat can be readily stripped. The lymph rapidly increases and forms a thick yellowish white layer on the viscera, especially at the upper part of the abdomen, and flakes in the fluid, the amount of which quickly becomes great. If the patient live long enough the turbidity of the fluid increases and finally it is purulent. As the adhesions get firmer and more numerous, some of the fluid becomes isolated in "pockets," so that at the autopsy a number of isolated abscesses may be seen. Life is rarely prolonged much beyond the formation of pus, but it has been known that the patient has lived long enough for a number of the abscesses just described to burst into the bowel. Occasionally the inflammation is so acute that the exuded lymph and fluid are tinged by extravasated blood. Three points must be mentioned to complete the description: the process is generally extremely acute, so that distinct evidence of peritonitis is visible to the naked eye at the end of a few hours; the peritonitis is often most marked close to the point at which infection of the peritoneal cavity took place, *e.g.* when due to rupture of a gastric ulcer it is usually most intense near the perforation, and here too the exudation of lymph is greatest, it being quite common to find a quantity of lymph glueing the stomach in the neighbourhood of the perforation to some adjacent organ, so that the perforation has become almost or quite closed; and, lastly, the peritoneal fluid may be contaminated by the contents of the viscus, rupture of which caused the peritonitis, thus there may be food from the stomach or intestine—I have known water-cress found in the peritoneal cavity—fæces from the intestine, urine from the bladder, or bile from the gall-bladder, but this is very unusual. The histological changes are the same as occur during the inflammation of serous membranes generally; the details have been admirably described by Mr. Durham, and he has pointed out that very frequently the changes are most active in the omentum. This is important, for a careful examination of it may be omitted at a post-mortem examination. Prof. Adami has shown how the omentum quickly applies itself over the wound of any abdominal viscus. From his and Mr. Durham's observations it is probable that the omentum is a more important organ than is usually thought.

All the above described appearances, indicative of acute general peritonitis, may be found restricted to particular areas. Such local conditions, *e.g.* perityphlitis, are described elsewhere in this work.

Some, but not many, sufferers from acute general peritonitis recover, but then the process is always or almost always arrested short of the formation of pus. Gradually the lymph becomes organised, new vessels grow into it, and fibrous adhesions binding together the viscera are formed. These adhesions may by their subsequent contraction drag upon or compress the intestine and cause constipation or even intestinal obstruction, or a piece of intestine may slip under a fibrous band and become strangled, or, again, these adhesions may, by their traction on the stomach, lead to various dyspeptic symptoms, among which pain is usually prominent.

Symptoms.—These vary so that perhaps it will be best to describe a typical case and then to point out the chief deviations. The following is a common sequence of symptoms. A young woman, who may have suffered from indigestion, but who is usually quite unaware that she has a gastric ulcer, is suddenly seized with abdominal pain so severe that she has to leave off what she is doing and go and lie down. The pain continues to be excruciating and sharp, so that she cannot keep still and cries out. It is usually felt at the front of the abdomen, and is often referred to the neighbourhood of the perforation. Quickly she begins to feel collapsed, her features are drawn, her pulse fails, the temperature rapidly drops, she becomes pale and covered with a clammy sweat, but remains conscious. The group of symptoms experienced so far are to be attributed to the perforation only.

Supposing a doctor sees her a few hours after the attack, he will find that she is still in great pain, which she probably describes as sharp or cutting, and which she says has spread from where it first began. It is greatly increased by any change of posture, by deep breathing, coughing or sneezing, or any movement of gas in the bowels. The abdomen is very tender, even the slightest pressure may cause agonising pain, and a cradle may be necessary to keep off the weight of the bed-clothes. Sometimes the tenderness is most in the region in which the peritonitis began. The skin is often hyperæsthetic. Because of the great pain caused by movement the abdomen is still during respiration, and the lower ribs move but slightly, consequently the breathing is short, rapid, and entirely thoracic. Often at this early stage there is no distension, but the abdominal muscles are very rigid, the abdominal wall is like a board, and it may be retracted even to such a degree that the front of the abdomen appears to rest on the spine. On the other hand, if the peritonitis have by this

time paralysed the wall of the gut, the abdomen is somewhat distended, the distension being usually first observed above the umbilicus; but whether distended or not, the abdominal wall is motionless. In order to keep the abdomen in the most comfortable position possible, the patient lies on her back with the legs drawn up, and it may be that to help the respiratory movements by the upper part of the thorax the arms are extended above the head. Her look is so peculiarly anxious that to the experienced observer it indicates severe abdominal disease. Her pulse is rapid and rather small; her temperature is subnormal, or it may have begun to rise. She complains of nausea, and perhaps already retching and vomiting are present; if the pain is very great she may be sweating. By the end of twenty-four hours she is usually very much worse. The abdomen is quite motionless, but is much distended and uniformly tympanitic; the pain and tenderness have not abated; the posture is the same, and the muscles are still rigid. She looks pinched, ashy, and much more anxious; her eyes are sunken and surrounded by dark areolæ, and she has the appearance of a person suffering from severe toxæmia. The pulse is about 110, small and rather wiry; the temperature varies much in different cases, but it is probably about 100; the character of the respiration is unaltered. The retching and vomiting persist, the tongue and mouth are dry, and she complains of much thirst; the bowels have not been opened, and micturition is difficult and painful. From this time onwards the pulse becomes more rapid and wiry; the temperature keeps between 100° and 102° or 103°. The breathing becomes more and more shallow as the distension of the motionless abdomen becomes greater and greater; the abdominal note is tympanitic, with some dulness in the flanks; the tongue and mouth become very dry; thirst is very distressing; retching continues; but little is brought up; hiccough has set in; abdominal pain and tenderness become less, especially if pus is forming; probably no water has been passed because of the pain accompanying the effort, but when a catheter is passed not much is drawn off; the bowels are still constipated. Finally, the prostration is profound; the face becomes more sunken, it is ashen and cyanotic, and the cheeks are quite hollow; the fingers are cold and clammy; the tongue and mouth become more foul; the pulse becomes very rapid, varying from 150 to 200; it is more and more thready, and at last, perhaps two or three days after the first onset of pain, the patient dies. The examination of the blood is of importance, for there is hardly any disease with a greater leucocytosis than virulent peritonitis; the

leucocytes may quickly amount to as much as 100,000 per c.mm., 90–95 per cent of the cells being of the polymorphonuclear variety; on the other hand, if the collapse be great throughout there may be no leucocytosis. Inasmuch as typhoid fever is one of the few specific fevers in which there is no leucocytosis, the onset of this phenomenon may be of great help in diagnosing whether perforative peritonitis has taken place.

Let us now consider some of these symptoms a little more in detail. The sudden shock in cases of rupture of a viscus may cause almost fatal collapse directly it occurs. On the other hand, the onset of symptoms is often more gradual than indicated above, *e.g.* in puerperal peritonitis, and in cases in which the formation of adhesions leads to a slow leakage rather than a sudden rupture.

The sunken eyes, the pinched, anxious, slightly dusky or slightly livid look, which is often called the "facies Hippocratica" or "facies abdominalis," are often absent, and the face may either show nothing out of the common, or it may be flushed. Often it is a mere ill-defined something about the patient which correctly enables the doctor to determine that she is very ill. But if the toxæmia be very severe the patient is clammy, covered with perspiration, her skin is a blue anæmic colour, and the fingers are cold, and it is remarkable how quickly the whole aspect of the patient changes: the cheeks become hollow, and the whole countenance looks ashy, pinched, and restless. The degree of abdominal distension is most variable; the abdomen may even remain retracted till the end, a severe retraction usually points to perforation of a viscus as a cause for the peritonitis. On the other hand, the distension may be enormous, the recti stand out, and the transverse lines show admirably; the hepatic dulness is diminished, and the heart is pushed up. Occasionally the abdominal wall moves enough for lymph on it rubbing against lymph on the liver or spleen to produce a rub audible to the stethoscope and palpable to the hand. The amount of fluid effused depends much upon the time the patient lives. Often she dies before it can be detected during life; but if she lives long enough there may be fluctuation, a thrill and dulness shifting with position, unless the fluid be held up by adhesions. If gas be present as well as liquid, manipulation may give rise to a splashing sound. The tongue is at first white and coated, but it soon becomes dry and brown; the breath is foul, and sordes collect on the teeth. The appetite is lost, but, as already remarked, thirst is very distressing, the patient complaining much of it and of a horrid taste in the mouth. Sometimes,

especially in puerperal cases, there is diarrhœa, sometimes the bowel may act almost naturally. It is by no means infrequent for the vomiting to be very distressful, and the matters brought up may have a fæcal smell. Towards the end of the case the regurgitation of a quantity of filthy brown fluid may be drawn into the lungs and so kill the patient. The mind is usually clear throughout, but towards the end the patient may be very restless, and toss about the bed. Occasionally she becomes delirious. Insomnia is very common, and is frequently associated with much restlessness.

The pulse is usually as described, but it may be more rapid. The respirations may reach 60 a minute. The temperature is very variable. When the effect of the toxin appears to be out of all proportion to the reaction it induces in more favourable cases, the temperature is raised but little above normal during any period of the illness, and if the temperature remain subnormal for as long as two or three days the outlook is very grave. In other cases in which reaction is present to a considerable degree the temperature is raised to 103°, 104° or even 105°, but in any case the course of the temperature is very irregular. Towards death it may amount to 106° or even more, or it may sink below normal. Rigors may be present at the commencement, but they are not common, and if they occur late usually indicate that the patient has a local collection of pus. On the other hand, she often complains of a feeling of chilliness. The sweating, especially towards the end, may be very profuse. The urine is scanty, concentrated, high-coloured, with an abundant deposit of urates.

Acute and even suppurative inflammation of the peritoneum sometimes produces few symptoms, especially if the patient be old or worn out by some serious chronic disease, as cancer or tubercle, in either of which even perforative peritonitis may occur and yet be unsuspected during life. Such patients have no pain, tenderness, distension, vomiting, or constipation, but the tongue is usually dry, the pulse very quickly becomes very rapid, and the extremities very cold, and the patient quickly dies. Some authors consider that restlessness is very conspicuous in the cases of so-called masked peritonitis, but it certainly may be absent. Large doses of morphia obscure the symptoms of peritonitis. Peritonitis of the new-born rarely gives rise to any symptoms which lead to its recognition; it may be due to infection from the umbilical cord or to rupture of the colon, the cause of which accident has not yet been made out.

The duration of cases of acute general peritonitis varies much,

and the reason is that, as the area of the peritoneum is extensive, and its capability for rapid absorption is great, if the toxine manufactured by the offending micro-organism be very virulent, the body is quickly thoroughly poisoned, and the patient may be dead in twenty-four or thirty-six hours. It has probably struck the reader that some of the symptoms given are those of toxæmia, *e.g.* the prostration, pinched face, the sweating, and the profound general depression, and in just the cases in which these symptoms are most evident the toxæmia is most severe, and therefore death is most rapid, and very little sign of peritonitis is found at the autopsy.

Among 77 fatal cases collected by Treves, in 14 per cent death occurred within thirty-six hours, in 6 per cent between thirty-six and forty-eight hours, in 20 per cent between the third and fifth day, in 33 per cent between the fifth and seventh day, and in 27 per cent after the seventh day. It is singular that the only complication of peritonitis is pleurisy, which is occasionally accompanied by pneumonia. It occurs in peritonitis produced experimentally in animals, and is met with in about 20 per cent of cases occurring in man. It is more common on the right side than on the left. Acute peritonitis never leads to any appreciable inflammation of the subjacent viscera.

Prognosis.—Acute general peritonitis is very fatal. Out of 100 cases occurring at the London hospital, death occurred in 77, and these figures probably understate the mortality, for it is quite likely that some of these cases that recovered were instances of partial, not general peritonitis. Children and old people do very badly, and perforative peritonitis is particularly fatal. Probably cases arising in the female pelvic organs do best. One case I saw which was almost certainly gonorrhœal did especially well, and those due to disease of the appendix without actual perforation do next best. The more grave the symptoms of toxæmia the worse the prognosis; the rate of the pulse is also a very valuable guide, and so especially is the rate of the disease. Often by the time we see the patient she has been ill twenty-four hours, and if by then the symptoms are already very grave there is little or no hope of recovery.

Diagnosis.—For the first few hours after perforation has occurred it may be very difficult to tell it from the pain of an acute strangulation of the bowel, hepatic, renal or lead colic, cholera, dysentery, ulcerative colitis, acute pancreatitis, or twisting of an ovarian pedicle, but most of these can soon be differentiated.

There are no particular rules to guide us. Mistakes are best avoided by extreme care in consideration of all possibilities, and it is particularly necessary to remember that few diseases vary so in their clinical aspect as acute general peritonitis. Many symptoms are often absent, and he who considers one symptom as essential is sure to be led into error. Even pleurisy at the base of the chest has been mistaken for peritonitis. Most often the problem before us is to determine whether intestinal obstruction is present or not, and the question is greatly complicated, because, as we have seen, strangulation of bowel renders possible the passage of bacteria through it, and therefore many cases of acute obstruction of the bowels quickly become complicated by peritonitis. However, the following points may help. Absence of pyrexia is in favour of intestinal obstruction, so also is rapid distension of the abdomen, especially if coils of intestine in which peristalsis is visible can be seen, but such coils have been seen in acute general peritonitis without obstruction. Early and urgent vomiting is in favour of obstruction, in which condition the pain is usually paroxysmal; the abdomen is not board-like nor tender; there is no cutaneous hyperæsthesia; the patient does not lie in the true attitude of peritonitis, but may bend forward, "doubled up" by the pain, and the abdomen moves with respiration until later in the disease than is the case with acute peritonitis.

Acute pancreatitis may give rise to much difficulty, but in that disease the symptoms are rather those of acute intestinal obstruction without any of the mechanical effects of it, such as distension.

Lead colic should not be forgotten. In this condition the pain is paroxysmal; there is no tenderness or hyperæsthesia, indeed pressure often relieves the pain, which, if it causes the patient to alter his position, leads him to bend forward instead of taking up the attitude described as characteristic of peritonitis. Vomiting is very rare, and there is no fever. A blue line is often present on the gums, and lead may be detected in the urine.

The diagnosis of the cause of the peritonitis is often very difficult. Obviously the history may point to perforation of a gastric ulcer, a typhoid ulcer, puerperal peritonitis, appendicitis or injury. A vaginal or rectal examination may be of great service. Very sudden onset with collapse and pain point to perforation as a cause.

It is excessively rare for the cause to be outside the abdomen. Peritonitis has been described as occurring in association with

specific fevers, especially erysipelas, scarlet fever, typhus, measles, small-pox, and influenza, but the occurrence is so exceptional that many authors are very sceptical. Certainly sometimes the peritonitis is really local, as in a case of typhus in which it was due to rupture of a splenic infarct. If peritonitis were present in these specific fevers and had not a local cause, it is highly probable that the symptoms of it would be overshadowed by those of the specific fever. Mention may here be made of rheumatic peritonitis. In all probability this disease does not exist. Many of the cases described in bygone days got well, so the diagnosis is open to grave doubt; it is almost certain that the peritonitis was really due to gonococci or pneumococci.

Nowadays the only two forms of peritonitis which we can expect to recognise clinically, and are not due to abdominal disease, are pneumococcal and that which occurs with Bright's disease. Max von Brunn collected a number of recorded cases of pneumococcal peritonitis, from which it appears that the disease is usually ushered in with very sudden rigors and the patient is quickly alarmingly ill. It appears, therefore, that if no other cause for peritonitis could be discovered, early severe rigors might suggest that it was pneumococcal, and it is interesting to note the similarity of symptoms produced by invasion of the lung and invasion of the peritoneum. Further, it is said that if the patient live long enough the temperature will fall quickly about the seventh day, as it does in pneumonia of the lung. After the first onset the symptoms become less urgent; diarrhœa is more common than constipation, and if the patient live long enough the purulent effusion usually becomes encysted, in which case it is easily evacuated. In most of the cases in which pneumococcal peritonitis has got well the abdomen has been opened. Obviously the presence of pneumonia would be a great help to diagnosis. The acute peritonitis, which may be the terminal event of Bright's disease, either acute or chronic, occurs in about 10 per cent of fatal cases of Bright's disease. Usually it gives rise to no symptoms, and therefore it is not commonly recognised. Most cases are purulent.

Treatment.—When called to a case of acute general peritonitis the physician must at once make up his mind whether or not to advise operation, for very quickly the time during which an operation can be performed with any hope of success passes away. Therefore, if it be decided not to operate it is well, as a rule, not to alter this decision. Unfortunately, in many cases by the time the

patient is seen all chance of recovery has passed away. The treatment of puerperal peritonitis hardly falls within the scope of this article, so it will not be considered in the remarks which follow. There is no evidence that perforative peritonitis can get well without opening the abdomen and closing the perforation, and therefore directly this diagnosis is made the case must be handed over to a skilful surgeon, and every hour lost greatly increases the patient's risk. While, on the one hand, the physician should use his very best judgment to prevent an unnecessary laparotomy, he must remember that perforative peritonitis is necessarily fatal unless the surgeon intervene, and, further, that if done properly the mere opening of an abdomen does very little harm, therefore, if the physician is in any doubt as to whether the abdomen should be opened a laparotomy should be performed. If perforative peritonitis be present the patient's life may be saved, and if it be not the danger to him is slight, provided, of course, that the operator is accustomed to such operations and is thoroughly versed in aseptic and antiseptic principles. In connection with this it should be remembered that although the symptoms of perforation of a typhoid ulcer are often indistinguishable from those of the collapse so common in the third week of typhoid fever, experience has shown that an exploratory laparotomy properly performed adds but little to the patient's danger, even when his condition is due to simple collapse, and therefore in all cases of doubtful perforation in the course of typhoid fever the abdomen should be opened. Cases of traumatic peritonitis should also be handed over to the surgeon. The physician, therefore, only has left very few, and they are either those in which because of the duration or the severity of the toxæmia the case is hopeless, or the milder cases, and these are almost always due to spread of inflammation from the appendix or the Fallopian tubes. Having for any reason decided against operative interference the following treatment should be adopted. The patient should be kept in bed absolutely at rest on his back, with, if necessary, a cradle to take off the weight of the bed-clothes, and a pillow under the knees to enable him to keep them flexed on the abdomen with comfort. Very little food is necessary, for the acute stage is so short that deprival of food is not of much consequence, and it is desirable to give but little by the mouth because of the sickness, and because the functions of the stomach are seriously impaired. Rectal feeding is much to be preferred, but it is often very badly carried out. The enema should be of such a temperature that when it reaches the rectum its temperature is 100° F. Usually at first the patient is too ill

to be moved, but later on if possible the buttocks should be raised and the patient very gently turned on the left, so that the enema will flow downwards by gravity towards the sigmoid, for it is much better absorbed and better retained if spread over a large surface. If it is left lying in the neighbourhood of the internal sphincter it is very liable to reflexly set up movements of defæcation. It should be given slowly, best with a funnel and a long soft tube passed very gently high up; the patient should lie with the buttocks raised for some time after the enema is given. Beddard has well pointed out that most nutrient enemata are unsatisfactory. Not only do they contain far too few kilo calories of energy, but much of the energy is due to fat which is very feebly absorbed by the rectum. Therefore a nutrient enema should consist of proteid and carbohydrate, but as it is quite impossible to get in enough of both, it is better usually to sacrifice the carbohydrate. A very good enema is somalose, $3\frac{1}{2}$ oz. = 240 kilo calories; dextrose, 5.8 per cent solution (this is isotonic with the blood), 9 oz. = 58 kilo calories = a total of 298 kilo calories. Starch is very readily absorbed, and an enema made of starch, the white of three eggs, and 9 oz. of water, in which 40 gr. of salt are dissolved to form an isotonic solution, is often useful. White of egg is absorbed even if not predigested, but milk is not. In order that the patient should be disturbed as little as possible the enema should be as large as possible, and often an enema of a bulk of 9 fluid oz. given four times in the twenty-four hours can be retained, but if this is not so the bulk should be less and the enema given more often. The rectum should be washed out with warm water once in twenty-four hours. Nutrient suppositories are almost valueless. Patients who are being fed per rectum get far too little fluid in their body, especially if they are vomiting, therefore half-a-pint or a pint of sterilised normal saline solution (a drachm of salt to a pint of water) should be injected once or twice a day into the subcutaneous tissue of the axilla at a temperature of 100° F. If any food is given by the mouth it should only be an occasional teaspoonful of milk, unless there is no vomiting. Vomiting is often very difficult to treat. The drugs which are most useful are dilute prussic acid in an effervescing mixture, and drop doses of tincture of iodine. Sometimes the sickness is less when the patient is taking opium, but this may increase it. If after two or three days it still continues, to the great distress of the patient, the stomach should be washed out; this may appear bold, but I never knew it do any harm in the cases we are considering, and it often gives great relief. Thirst is very distressing; it is best relieved by a

rectal injection of half-a-pint of warm normal saline solution; if the patient craves to have something by the mouth he should be allowed a little milk or water, cold or warm as he prefers, even if he be sick after it, and this is generally better than beef-tea or such things. It is a common mistake to give far too much ice to suck; a large quantity of iced water in the stomach greatly increases the general depression. The mouth should be kept clean; a weak mixture of tincture of myrrh and water is as good a wash as anything, and ten grains of borax to the ounce with a little glycerine may be added to it.

The drug which does most good in peritonitis is undoubtedly opium; it relieves the pain, keeps the bowels still, and quiets the restless patient, and gives him what is most desirable, namely, sleep. The student nowadays is likely to be confused, for he hears surgeons saying, withhold opium, and he reads books of thirty years ago strongly urging it. So long as it is likely that an operation will be performed for the peritonitis, the opium should only be given so as just to relieve pain, but when once it has been decided to try to get the patient well without operative interference the opium should be steadily given, so as just to keep the patient slightly drowsy. The gain is immense; the exhaustion due to the pain, restlessness, tossing about, and insomnia are all prevented. Inasmuch as the breathing is hampered by the state of the abdomen, it is not much use trying to observe the effect of the opium upon it, but the pupils should be watched. Probably the best way to give it is simply to add enough laudanum to each nutrient enema; if they are given to an adult every eight hours, fifteen or twenty drops of laudanum may be added to each, but the amount required in each patient varies immensely. For the acute pain at the beginning of the illness an injection of morphia is often necessary. There is no need to give more opium than is necessary to control the above-mentioned symptoms, and therefore the quantity may usually be soon diminished. If there be no sickness it may be given by the mouth. If it produce any unpleasant symptoms a little belladonna may be mixed with it, but this is usually undesirable, as the mouth is already very dry. Much has been said about the desirability of giving purgatives in peritonitis, but that only refers to certain conditions arising after operation. They are quite out of the question in the cases under consideration.

SIMPLE CHRONIC PERITONITIS

Most cases of chronic peritonitis are due to either tubercle or cancer, but there are a certain number in which, as far as we can discover, neither of these causes exists, nor can the immediate determining cause be discovered. Such cases are in England called simple chronic peritonitis; in Germany it is usual to speak of them as idiopathic. It has been suggested that they are all really tuberculous, but that in "simple chronic peritonitis" the fibrous tissue is abundant, and the tubercle bacilli are few; but I have not noticed in post-mortems that I have made anything in the character of the peritonitis, or in the condition of the various organs of the body to support this view.

The disease is fairly common; it is met with about equally in the two sexes, and is very rare in children, but in adults it occurs at any age.

Morbid anatomy. — The peritoneum in these cases is thickened and opaque white. There may be adhesions which may mat the whole intestines into one mass, or may form loculi in which fluid collects, but the presence of adhesions is by no means so constant as in tuberculous peritonitis. The omentum is particularly liable to be puckered up and form a thickened mass lying transversely across the abdomen just above the umbilicus, and the mesentery nearly always shrinks and draws the intestines back towards the spine; often two irregular lumps of thickened peritoneum may be felt in different parts of the abdomen. The effused fluid is usually pale straw colour, but rarely, if the patient have chronic Bright's disease, it may contain blood, but it is never purulent unless the abdominal cavity has become infected from tapping.

The peritonitis is nearly always particularly evident over the liver and spleen, indeed universal perihepatitis, in which the liver is covered by a thick white jacket which easily peels off (*Zuckergussleber*), is nearly always only a part of a general simple chronic peritonitis. Thus I took quite at random twenty-two examples of complete perihepatitis from the post-mortem records of Guy's Hospital, and I found that in only two was it stated that there was no peritonitis, and in seventeen it was definitely stated that chronic peritonitis was present. One description taken gives a very good idea of simple chronic peritonitis:—"The peritoneum was converted into a thick tough layer, matting all the intestines together and

covering them with a thick fibrous apron. The omentum was all matted up into a thick lump two inches broad." Chronic Bright's disease is often found in those who suffer from simple chronic peritonitis. Sometimes the disease is associated with alcoholism, sometimes with syphilis, and occasionally with long-standing backward pressure from disease of the heart or lungs. It is not uncommon to find after death chronic pericarditis and chronic pleurisy as well.

Symptoms.—The chief symptom is ascites. There is no other disease—unless we except very rare cases of colloid disease—in which so much fluid is poured into the abdomen as in simple chronic peritonitis. I have had a patient under my care in whom 790 pints were poured out; he was tapped thirty-four times; the largest amount removed at any one tapping was 31½ pints, and it appeared that sometimes the fluid re-collected at the rate of over 2 pints a day. It is most important to remember that in any case of ascites, if two or more tappings be necessary, that it is in the highest degree probable that the cause of the ascites is simple chronic peritonitis, and that even if, as is often the case, cirrhosis of the liver be present also, the ascites is to be assigned to the chronic peritonitis rather than to the cirrhosis. The patient shows the usual physical signs of ascites, save in the rare instances in which the fluid is loculated; the umbilicus may be prominent and the thickened omentum and other peritoneal thickenings may be felt, especially when the fluid has been recently withdrawn, and as the shrinking mesentery drags the intestines back, it is in these cases that a comparatively small amount of fluid leads to dulness all over the abdomen. The patient is usually thin, pale, and sallow; he suffers from constipation and indigestion; he gradually wastes; his skin becomes dry and wrinkled; towards the end diarrhœa often supervenes, and he usually dies of exhaustion. The symptoms of the disease with which the chronic peritonitis is associated will be present also. The outlook is very grave, but occasionally patients recover completely, and even when the case appears to be severe.

The best **treatment** is to tap the abdomen from time to time, as the ascites calls for tapping. Considering that simple chronic peritonitis may possibly be due to syphilis, it is well to give a prolonged course of large doses of iodide of potassium, and to apply a binder smeared with mercurial ointment over the abdomen. Diuretics should certainly be tried, and of these there is none better for ascites than 15 grains of copaiba resin suspended in mucilage, and taken three or four times a day; but, unfortunately, this is very

nasty, and often upsets the stomach. If that be so, a mixture containing digitalis, scoparium, and caffein forms an excellent diuretic. The well-known pill containing a grain of each of powdered digitalis leaves, powdered squill, and blue pill, is also often efficacious. Purgatives, especially those, like compound jalap powder, which cause a watery evacuation, may be useful, but they must be given with great caution, for these patients are very liable to diarrhœa, which may prove fatal, and also they must be given on an empty stomach, otherwise food may be swept away by them, and so be useless to the patient. It is very important that by judicious feeding the patient should take as much food as possible, and he ought to have plenty of fresh air.

The only variety of peritonitis that does not come under the heading of acute, tuberculous, malignant, or simple chronic, is that due to actinomycosis, but this is so rare that it hardly calls for description here. The older authors described a rheumatic peritonitis, but there is no convincing evidence that it occurs. General peritonitis may be very occasionally met with in the new-born. In most of these cases it is due to infection from the umbilical cord.

Local peritonitis is very common, but it is best described as a disease of the organ, *e.g.* the appendix, from which it spreads.

TUBERCULOUS PERITONITIS

Etiology.—Tuberculous peritonitis is due to infection of the peritoneum by the tubercle bacillus. It is easily produced in animals by the injection of tuberculous sputum into the peritoneal cavity. By the time there is an opportunity of examining the case in the human subject the infection is certainly often mixed. Statistics vary very much as to the relative frequency in the two sexes. Certainly more cases are operated upon in women than in men, but some statistics from the dead-house indicate that the disease is common in men. The point is not of much importance, as the disease is common in both sexes. Statistics also differ greatly as to the ratio of cases of tuberculous peritonitis to all cases in the dead-house. The highest figures are those of Boischke, who, among 4250 autopsies, found tuberculosis in 1393 (32.8 per cent), and in 226, or 16 per cent of these 1393 cases, the peritoneum was affected, and Nothnagel says the percentage varies in different collections of cases from $1\frac{1}{4}$ to 16. Locality probably has something to do with the difference. Primary tuberculous

disease of the peritoneum is generally considered to be rare, according to most authors not occurring oftener than one case in fifty of tuberculous peritonitis. But usually so many other organs are affected by the time that an autopsy is made that it is difficult to decide upon the primary seat, and Wilks and Moxon doubt whether primary affection of the peritoneum is after all so very rare. One primary seat, however, that is especially noteworthy is tuberculosis of the female genital organs. Sometimes, too, it occurs in association with tuberculosis of the prostate or vesiculi seminales. The lungs and intestines are very frequently affected, but it is usually impossible to say which was the primary seat. Sometimes this is to be found in tuberculosis of bones or of mesenteric or bronchial glands. The peritoneum is probably infected by the blood. The disease is most common between the ages of twenty and forty, thus Bottomley, collecting 538 cases, found the ages as follow: under ten years, 30; between ten and twenty, 112; between twenty and thirty, 137; between thirty and forty, 122; between forty and fifty, 93; between fifty and sixty, 31; between sixty and seventy, 11; and over seventy, 2. The rarity of the disease under the age of ten is a strong argument against the supposition that infection takes place directly from the intestine as a result of the ingestion of tuberculous food.

Morbid anatomy.—The conditions found at death vary very much, but every one must be struck with the fact that tuberculous peritonitis is rare, although subperitoneal tubercles in connection with tuberculous ulcers are so common. In the simplest form all that is found are a number of gray tubercles of varying sizes, but none bigger than swan-shot, scattered over the peritoneum, especially under the diaphragm. There are usually very few over the intestinal peritoneum. In these cases much slightly turbid fluid is secreted, so that the abdomen may contain several pints, but the adhesions are few. In rarer cases in which there is little or no fluid there are a few adhesions composed of soft lymph. In the most common form of tuberculous peritonitis the most striking thing is the enormous mass of adhesions; all the abdominal organs may be taken out of the abdomen as a solid whole as though they had all been fixed by having plaster of Paris run into the abdominal cavity, but the extraction of them is not easy, for there are many adhesions to the abdominal wall. I have in such a case spent a couple of hours trying to dissect out the intestine from the swollen mass, and have then given up the task as hopeless, finding every coil of intestine tightly fixed to other

coils, so that when a cut is made across the whole mass a number of sections of the gut are seen, and they do not collapse when they empty, for they are held open by adhesions. The intestinal walls are much softened, so that they are torn in trying to get the intestines out or to separate them. In among all these adhesions are numerous cavities containing fluid, which may be purulent, and these abscesses commonly discharge themselves into one or more adjacent pieces of intestine, so that all sorts of fistulous communications are formed, and sometimes such a collection discharges externally on the one hand and into the bowel on the other, so that there is a fæcal fistula opening externally. The liver, kidneys, and spleen are firmly adherent to the mass of intestines. In cases in which the adhesions are less marked, although extensive, the omentum is commonly puckered up and thickened, so as to form a transverse tumour lying across the abdomen just below the transverse colon, and various other masses of thickened peritoneum occur in various parts of the abdomen. The peritonitis may be entirely fibrous, but caseous masses are commonly present, and the mesentery is often puckered, so that the intestines are drawn back to the spine. Usually not much fluid is present in these cases, and it is commonly localised. It varies in consistency, but it is usually turbid and not rarely purulent. It may contain blood. The abdominal lymphatic glands are usually caseous, but it is an error to suppose that the enlarged caseous mesenteric glands are often felt during life. When masses supposed to be these glands are felt, it nearly always turns out that they are really hard masses of omentum or other peritoneum affected by tuberculous disease. Tuberculous disease may, of course, be found in other organs, and tuberculous peritonitis is often associated with cirrhosis of the liver.

Symptoms.—In rare cases the disease may fairly be called acute, but it is then only part of a general acute tuberculous process, and there are rarely any signs to indicate definitely that the peritoneum is affected. At the post-mortem it is found studded with gray tubercles, and some fluid is free in the peritoneal cavity.

The symptoms of chronic tuberculous peritonitis may be very vague. It is commonly thought to occur chiefly in children, but the figures already given show that this is an error which probably arises from the fact that in children many cases of tuberculous ulceration of the bowels are incorrectly styled tuberculous peritonitis. Another point to be borne in mind is that although tuberculosis of the peritoneum is rarely primary, it is by no means

uncommon for it to be the only evidence of tubercle detectable during life.

By the time the patient is first seen he has usually been out of health for some weeks; he has felt languid; his appetite has been poor; he has suffered from various symptoms of indigestion and his bowels have been irregular; he has been liable both to constipation and diarrhœa; he may have complained of abdominal pain, and if the patient be a child he has probably been sick. The doctor on examining him will notice him to be pale; the tongue is a little furred, and the abdomen is probably distended. His pulse will be slightly accelerated, and the temperature may reach 100° every evening and there may be night sweats. Slowly the patient becomes worse, the pallor increases, he loses flesh, the gastro-intestinal symptoms continue, the abdomen becomes more distended and feels turned, signs of fluid can be detected, but most of the swelling is tympanitic, a hard mass of thickened omentum can be felt running across the abdomen just above the umbilicus, and other lumps may be felt in other parts, and the abdomen may be tender. The umbilicus is protruded and thickened, and may be abnormally red, the evening temperature is higher and the patient has lost weight. Gradually he loses ground, the dyspeptic symptoms increase, the tongue becomes dry and fissured, and finally he is seen to be generally anæmic, very thin, and wasted; consequently his veins show easily, his skin is dry, wrinkled, and brown, the face is sunken, but in striking contrast to the large distended abdomen. The feet may be œdematous from the severe anæmia. If the patient has reached this stage he usually slowly sinks and dies. Such a case as this corresponds to what has been called the fibro-caseous variety. We will now consider the symptoms a little more in detail. A recollection of the morbid anatomy will show that the state of the abdomen must be most variable. It may be retracted, or it may be so distended that it appears likely to burst. The fact that the fluid is often localised by adhesions may make fluctuation difficult to obtain; on the other hand gurgling may be felt on pressure as fluid is driven from one loculus to another, and some days the fluid may be more easy to detect than others. Also it is more in the front of the abdomen than in most cases of ascites, because shrinkage of the mesentery draws the intestines back. The thickened omentum and other lumps are not felt with equal ease on following days, probably because their successful palpation depends much upon the amount and position of the gas in the intestines and fluid in the abdomen.

The whole abdomen often has a doughy feel. The redness of the protruded umbilicus is by no means constant, but is a valuable sign when it is present. The protruded umbilicus may contain fluid that can be pressed back into the abdomen. There may be a fæcal fistula, and if so it is at the umbilicus. The peritoneal inflammation may give rise to difficulty of micturition.

The tongue may become very dry and fissured and the mouth foul. The appetite is most variable; it may be lost or voracious, with a special liking for curious articles of food; constipation usually alternates with marked diarrhœa, and the motions are commonly foul. Vomiting, nausea, and flatulence are common. The temperature is most variable in the same case, and among different cases sometimes it ranges about 103° or 104°; at other times it is normal or subnormal, but it is usually higher in the evening than the morning. The pulse is feeble and rapid, wasting and weakness are very striking, and few things are more characteristic than the extreme general wasting, in contrast to the distended abdomen.

The above represents the usual picture, but sometimes we meet with cases which are characterised by but little change in the peritoneum, although much ascitic fluid is effused. This is commonly known as the ascitic type, and it is to be borne in mind that tuberculosis of the peritoneum is by far the commonest cause of ascites in children. These cases are frequently associated with pleurisy, and, according to Marfan, the febrile symptoms soon pass away, the pleurisy subsides, and the case becomes one of chronic ascites, with symptoms of disorder of the gastro-intestinal tract. Sometimes we meet with cases, known as the fibrous type, in which the formation of fibrous tissue is excessive, while but little fluid is effused. In these cases, owing to the shrinkage of the mesentery, the abdomen is retracted, and because of this and because the patient is wasted the bony prominences, as the crest of the ileum, stand out very markedly and the spine can be easily felt. This fibrous tissue compresses the intestine, so that these patients complain much of constipation, and they may be admitted to the hospital for the first time as cases of intestinal obstruction. The examination of the ascitic fluid in any case of tuberculous peritonitis rarely shows the presence of tubercle bacilli.

From this it is seen that the various forms of tuberculous peritonitis may be thus classified:—

Acute.

Chronic—1. Fibro-caseous. 2. Ascitic. 3. Fibrous.

But it must be remembered that there are many cases which

form connecting links between the groups, and the same case may at different times be in different groups; thus, if the ascitic form do not recover, it usually passes into the fibro-caseous form.

Complications.—It would lead us too far afield to describe the results of the tuberculosis of other organs which is so frequently associated with tuberculous peritonitis; here need only be mentioned the following:—The fæcal fistula forming at the umbilicus, which has been already described; this is much more common in children than in adults. Fistulous communications may form between various adjacent pieces of intestine, but they are generally only diagnosed when the communication takes place between the upper part of the jejunum and the colon; then the patient wastes rapidly and undigested food is found in the fæces; this condition is called "lientery." Allusion has already been made to the chronic intestinal obstruction associated with the fibrous form. Occasionally it is due to a band or to kinking of the intestine, but these are both rare causes of intestinal obstruction following tuberculous peritonitis compared with the frequency with which it is due to compression of and traction upon the intestine as a result of contraction of the new-formed fibrous tissue.

Diagnosis.—This may be difficult in the early stages, but general ill-health, if combined with abdominal pain, should always excite our suspicion, especially if the patient is a child. Occasionally it may be difficult to distinguish typhoid fever, but generally some sign of this will help us; in tubercle the morning temperature may be higher than the evening, this is not so with typhoid. Later on the difficulties nearly always take one of two forms, either to distinguish correctly the cause of the ascites, remembering that if encysted then tuberculous peritonitis is likely, or to distinguish the cause of a lump or lumps felt in the abdomen. It is to be borne in mind that the puckered, thickened omentum forms a transverse band across the abdomen just above the umbilicus, and that such a band may be mistaken for the edge of the liver, the greater curvature of the stomach infiltrated with growth, or fæces in or tumour of the transverse colon.

Prognosis.—It is extremely difficult to foretell the outlook in these cases, for at any time serious tuberculous disease of other organs, *e.g.* the meninges or the lungs, may prove fatal. Severe gastro-intestinal symptoms and severe toxæmic symptoms, such as a high temperature, a feeble pulse or much sweating, make the outlook very gloomy. Cirrhosis of the liver, history of heavy drinking, the presence of syphilis, the formation of fæcal fistula,

either external or internal, and loss of weight, all render the prognosis bad. The prospect is most hopeful in the purely ascitic form.

Treatment.—There are two distinct modes of treatment. The first consists of two parts—general treatment and local medication. The general treatment is such as should be employed for any tuberculous process. The patient should spend his time in fresh air, perhaps preferably by the sea; he should be in such a place that it is possible to lie out of doors all day, and at night sleep with the windows wide open. By the careful selection of appropriate articles of food, and by, if necessary, giving medicines to improve the condition of the gastro-intestinal tract, he should be encouraged to eat abundantly. Usually it is found that milk and farinaceous foods are the most suitable, and it is much better to give small feeds at frequent intervals than a few large meals, for these may provoke vomiting and diarrhœa. In England the most usual local treatment is the application of mercury by the smearing of some mercurial ointment on a large piece of lint which is kept applied to the abdomen by a flannel binder. The oleate of mercury is an excellent preparation for the same purpose, but the pharmacopœial 10 per cent is best diluted. In France the abdomen is treated with iodine and tightly bound.

The second mode of treatment is the removal of the ascitic fluid. This may be done either by simple puncture with a Southey's tube or a larger trocar, or by laparotomy. Simple puncture appears to suffice in a few cases, but laparotomy is much more reliable. If the fluid be in the general peritoneal cavity the abdomen is opened in the middle line; if localised the incision is made over the largest and most accessible collection. Some surgeons wash out the abdominal cavity with boiled water; others with weak antiseptics not likely to cause general poisoning; others touch the visible tuberculous nodules with iodoform; others remove any visible caseous masses that can be easily taken away; others excise the Fallopian tubes if these are tuberculous, but many get excellent results by simply letting out the ascitic fluid, therefore this seems to be the essential part of this treatment. Why benefit should follow is entirely obscure. Many suggestions have been made, but as there is nothing to support any of them they need not be considered here.

Two or three practical points are noteworthy about this operation. The intestines are often adherent to each other and the abdominal wall, and they very easily tear, therefore the adhesions should be torn as little as possible, and if they are torn the greatest

care must be exercised. Perhaps the chief drawback to the operation is the liability to rupture of the intestine with the formation of fæcal fistulæ. The abdomen should be sewn up after the operation. Experience shows that no better results follow drainage, but fæcal fistula is likely to succeed it. Again, because of the undesirability of interfering with adhesions, the Fallopian tubes should not be removed unless their removal is very easy, nor should caseous glands or caseous masses be taken away. There is no evidence that such procedures are of any striking benefit, and probably flushing and sponging of the peritoneum are quite unnecessary. The mortality from the operation alone is between $2\frac{1}{2}$ and 3 per cent. Tuberculosis of the wound does not occur. Sepsis is not so likely to follow as after operations on the healthy peritoneum.

We now come to the question, whether or not laparotomy should be performed. It certainly should not when the tuberculous peritonitis is merely part of a general acute miliary tuberculosis, nor should it be undertaken when fæcal fistulæ are already present, for in such a case the adhesions are so numerous that but little fluid can be let out, and separating the adhesions will further tear the bowel. The best results follow in cases in which there is a large amount of ascitic fluid free in the peritoneal cavity. It is true that these are just the cases which give the most favourable results when treated without the interference of the surgeon, but, on the other hand, they are the cases in which the operation is easiest, and therefore as the mortality, as a result of the operation, is in all cases so low, laparotomy should be performed on these cases if circumstances prevent fresh air and abundant food, or if these having been tried the progress is slow. Probably the cases in which surgical interference is most valuable are those in which there is much ascites and some fibro-caseous change in the peritoneum, but the adhesions are not numerous or dense enough to prevent the evacuation of the fluid. The outlook is poor when there is no ascites but much fibrous change leading to dragging back and binding down of the intestine. In such cases but little good is to be expected from laparotomy.

When tuberculous peritonitis is localised the same principles hold; if there be a large collection of fluid incision does good; if the result of the disease have been to form chiefly dense adhesions incision will probably do more harm than good. Mistakes are often made in the treatment of the umbilical conditions. There is a great temptation, when a fluctuating swelling appears at the umbilicus, to cut into it, but it is better left alone. It is nearly always a

localised tubercular abscess, and if left to itself may burst into the bowel, a much better result than its external rupture, for experience shows that sooner or later, even if opened externally, it will form a communication with the bowel and we shall have a fæcal fistula; but if left alone we may have no external opening, only a passage from one part of the bowel to another. Fæcal fistulæ that have formed at the umbilicus should be left alone; experience teaches that operative interference usually makes matters worse.

The treatment of the intestinal obstruction that follows the fibrous form does not come within the scope of this article.

Turning now to the results of the laparotomy. The immediate effect is nearly always an improvement in the patient's condition, but this is but little guide as to whether the improvement will be permanent. Not infrequently there is for some time after the operation a complaint of abdominal pain, which may persist for many weeks. The best result that follows is for the temperature to fall considerably after the operation and then gradually to come to normal, for the appetite, digestion, and general condition to improve, for the anæmia to pass off, for a gain of weight to take place, and for the patient to appear ultimately in excellent health. It need hardly be said that such a result is best gained if after the operation he be sent away to be in the open air and sun, and be carefully fed. It has occurred that in such cases it has been necessary later to open the abdomen again, because there was some protrusion of bowel from the wound in the abdomen, and then the peritoneum has appeared normal, and as far as the naked eye could detect the cure has been perfect. Sometimes the fluid re-collects and then the patient is cured after a second laparotomy. In other cases, although he can hardly be said to be cured, he is so much better after the operation as to completely justify its performance.

Several cases are on record in which there has been much improvement in tuberculous processes in the lungs, bones, or elsewhere after laparotomy for tuberculous ascites.

MALIGNANT DISEASE OF THE PERITONEUM

Etiology.—Usually when malignant disease of an abdominal viscus extends to the surface of the organ in which it grows it causes some local peritonitis, or some portion of peritoneum lying in contact with the malignant growth may become directly infected,

as, for example, when the free end of the omentum lying against a carcinoma of the uterus or ovary has developed in it a nodule of growth at the point of contact, or pieces of growth from some viscus may become detached into the peritoneal cavity and grow where they happen to lodge. Thus in a case of carcinoma of the liver nodules of growth may be seen after death, and even felt per rectum during life, in Douglas's pouch. But if the disease of the peritoneum extend no farther than above indicated, it is rare for it to give any signs by which it can be detected during life.

Malignant disease of the peritoneum, sufficiently advanced to be of considerable clinical importance, is either primary or secondary. Primary malignant peritonitis is very seldom seen, and if we except a very rare form in which a number of little nodules are found over the peritoneum, which is not recognised until after death, primary malignant peritonitis resembles the secondary form.

Secondary malignant peritonitis may follow upon malignant disease anywhere, but usually the primary form is in the abdomen, and most often in either the pelvic organs or stomach. The frequency with which it follows disease of the pelvic organs explains its comparative prevalence in women. Thus Fagge found that among forty-five consecutive cases of extensive malignant disease of the peritoneum, in only six were all the abdominal viscera free from disease, in nineteen the ovaries were affected, in seventeen the stomach, in three the uterus appeared to be the primary seat, and in two the rectum. Often it appears that the disease spreads, so that when the peritoneal surface of an organ becomes affected, it directly affects the peritoneum in contact with it, although no adhesions form.

Morbid anatomy.—The nodules of peritoneal growth usually appear most numerous in the flanks; individual nodules are often umbilicated. There is often much chronic inflammation in connection with the growth, and the affected peritoneum is very inclined to be puckered, hence the omentum forms a thick mass usually lying transversely across the abdomen, or below the right lobe of the liver; the mesentery may become shrunken; lumps may be felt in different parts of the abdomen, and puckering may so affect the hollow viscera as to cause obstruction. Considerable amounts of ascitic fluid are usually poured out, and commonly this fluid is bloody. Adhesions often lead to the collection of fluid in loculi. The carcinoma may be of any variety. In a very interesting, but rare group of cases,

the growth undergoes colloid degeneration. On opening the abdomen large gelatinous masses of colloid carcinoma may be seen; the omentum is a gelatinous mass, and the subserous coat of the viscera may be so full of this gelatinous material that the organs seem to be embedded in it. On section the new material is seen to be almost white and to be in a fibrous network. It is not always gelatinous, but may be thin enough to flow when the abdomen is tapped.

Symptoms.—If there be no symptoms to point to a primary source for the disease the diagnosis is difficult. The chief symptom is usually ascites, and it is found that commonly the fluid removed is not bloody; this is a great help to diagnosis, but it must be remembered that peritoneal fluid may be sanguineous in tuberculous peritonitis and in Bright's disease. The mass of thickened omentum and other masses are frequently felt through the abdominal wall. Sometimes secondary nodules of growth may be felt under the skin and the umbilicus may be implicated, and in some, at least, of such cases the growth arrives at the umbilicus by spreading up the obliterated umbilical vein. Pain may be present, but it is often absent. Constipation is common. As a rule the patient wastes and loses ground rapidly, the usual symptoms associated with cancer becoming quickly marked, but the course of colloid carcinoma may be very slow.

Treatment.—This can only be palliative. The fluid may be removed from the abdomen if the distension be great. It is well to first take away a sample with a fine needle, for if it be very bloody it should be removed very cautiously if at all.

Sarcoma, fatty tumours, lymphomata, and hydatids of the peritoneum really grow from the subperitoneal tissue, and are, therefore, more properly described under the head of retroperitoneal growths.

PERITONEAL SANGUINEOUS CYSTS

From time to time — although rarely — most physicians or surgeons of wide experience meet with cases of large cysts within the abdomen, which are not hydatid or cystic sarcomata, but which contain a brown or reddish brown fluid in which are found altered blood pigment, altered red and white blood corpuscles, blood crystals, and sometimes large cells probably derived from the cyst wall. To these some authors have given the unfortunate name of pseudo-cysts. Inasmuch as often the history clearly shows that the

cyst followed an injury, it is very likely that these cysts are due to the effusion of blood from a ruptured vessel. This view is supported by the fact that the cyst may appear quickly after a blow on the abdomen, as in the case of a man who was thrown down, and the man who threw him knelt on the abdomen, and by the fact that a large collection of blood, which probably would, had the patient survived, have formed a cyst, has been found after an injury between the layers of the transverse mesocolon. Sometimes the cyst has not appeared till long after the injury, when we may suppose that ulceration had eaten into a vessel; sometimes, again, the fluid seems to be spontaneously absorbed, only again to be effused.

These cysts are found much more commonly at the upper part of the abdomen than elsewhere, and usually they are in or near the middle line. In some the blood is effused into the lesser peritoneal cavity, and for some reason we do not know, it does not escape from the foramen of Winslow; it may in such a case pass into the great omentum. Then there is a large tumour, which becomes quite superficial towards the front of the abdomen, although it reaches to the back of the abdomen; it is tense, rounded, smooth, extends downwards to a little below the umbilicus and upwards nearly to the ensiform cartilage. Its lateral limits vary in different cases, and so does its size, for, as in many parts of the lesser peritoneal cavity, the surfaces are usually in contact, the effused blood may not at first separate them, and then probably adhesions quickly limit it. Such a tumour presses up the stomach, appearing from its lower border; it lies over the colon, which, together with more or less of the intestines, is pressed to the back. From their position these cysts are often regarded as arising from the pancreas, and as the patient usually completely recovers, it is generally impossible to certainly make out the origin of the cyst. Sometimes it may be that the pancreas is damaged, and blood is effused from it into the lesser peritoneal cavity, but if the cyst were a genuine pancreatic cyst it would not come into the great omentum, and would therefore be less superficial, having the stomach in front of it and the colon below it instead of behind it. Possibly in some cases distension of the stomach and colon with gas would help to a diagnosis. The facts that the fluid will convert starch into sugar and contains trypsin are strong evidence that the cyst is pancreatic, although either might be owing to a torn pancreas having led to effusion of blood into the lesser cavity, but then the fermentative power of the cyst fluid will be slight,

Again, many cases of genuine pancreatic cyst do not show the chemical characters of pancreatic fluid. Some genuine pancreatic cysts are undoubtedly blood cysts, and, on the other hand, it is probable that after a time the fluid of blood cysts may alter so as to make their origin obscure. It is extremely likely that many cases which were really blood cysts in the lesser peritoneal cavity have been recorded as pancreatic cysts, for, as already indicated, diagnosis between the two may be extremely difficult. Those very rare cases of cysts of the suprarenal body or cysts of the posterior wall of the stomach may easily be confounded with these sanguineous cysts of the lesser cavity.

Sanguineous peritoneal cysts may occur not only in the lesser peritoneal cavity, but also in the mesentery in the ascending, transverse, or descending mesocolon, and in the small omentum. Blood cysts, which may be difficult to diagnose from these, may spring from the retroperitoneal tissue.

These cysts may occur at any age and in either sex; often the history of injury is quite distinct; at other times no history of injury can be obtained. The rate at which they form, and the time after the injury when they are first seen, are not constant. Usually they are large and may contain several pints. They are smooth, dull, tense, rounded. If in the upper part of the abdomen they can usually be separated from the liver; there may be a band of resonance between the liver and the cyst, or the hepatic edge may be felt to move up and down with respiration, while the cyst, which often has a transmitted pulsation from the aorta, is fixed. The stomach above the tumour can be mapped out by percussion. A hydatid thrill may be obtained, and the bile duct has been so pressed upon as to lead to jaundice. The patient usually complains of attacks of colicky pain, often very severe; vomiting is frequent. These symptoms may come on very soon after an injury, but more frequently days or even weeks intervene. In some cases the vomiting and pain have continued for weeks, or even months, but have disappeared when the tumour has been large enough to attract attention. Having reached a certain size the tumour becomes stationary, and although vomiting and pain may have ceased, it still appears to affect the patient's health injuriously, since wasting associated with a sallow complexion is common. These general signs are the same whatever the position of the tumour, and it is to be remembered that those in the lesser cavity of the peritoneum often do not occupy the whole of it, and indeed sometimes they are confined to part of the cavity of the great omentum.

Most of the recorded cases have been operated upon, but in one case the cyst was known to have been present for twelve years when the sufferer died of cholera. Rupture of the cyst into the peritoneal cavity is very rare, but it may undergo spontaneous cure by rupture into the intestine. The cyst wall is fibrous and of very varying thickness; it may have an epithelial lining and minute hæmorrhages may be seen dotted under this. Considering the difficulty of a certain diagnosis it is probably always wise to open the abdomen when one of these cysts is suspected. Aspiration has often led to a complete cure, but occasionally this has not resulted till a second aspiration. Excision of the cyst and the injection of fluids into it both appear to be dangerous. Some surgeons have stitched the edges of the cyst to the abdominal wall and drained it.

RETROPERITONEAL SUPPURATION AND TUMOURS

It is usual, under this heading, to describe only suppuration in or tumours arising from the retroperitoneal tissue, and the lymphatic tissues therein. Diseases of the kidneys, suprarenal bodies, spine, bones, and vessels, although strictly retroperitoneal, are described elsewhere.

Pus in the retroperitoneal tissue is not uncommon. It arises from disease of some organ in its neighbourhood, and most often follows diseased bone or disease of the appendix, an abscess in connection with which is usually completely shut off from the peritoneal cavity, but may work its way into the retroperitoneal tissue. More rarely retroperitoneal suppuration follows perforation of some part of the bowel that is not covered by peritoneum. Formerly the suppuration in the loose connective tissue around the right kidney that may follow disease of the appendix was called "perirenal abscess," and was regarded as a distinct disease. Pus in the retroperitoneal tissue may form a very long tract for itself, thus the pus of an abscess which began in the appendix may find its way under the diaphragm and cause either suppuration of the mediastinal tissue or an empyema.

The diagnosis of retroperitoneal suppuration is not usually difficult. General signs, such as leucocytosis, a raised temperature, often markedly hectic, a quick pulse, loss of flesh and obvious illness, are nearly always present; in addition local tenderness and pain are common, and further it is often possible to feel a distinct tumour corresponding to the abscess. The treatment is entirely

surgical, but, inasmuch as the abscess can be drained without opening the peritoneal cavity, it is usually successful.

RETROPERITONEAL TUMOURS are so rare that their relative frequency cannot be stated, but by far the most important are the *sarcomata*, which are primary in retroperitoneal structures, but these are very infrequent; for instance, out of 14,630 tumours, of which 894 were sarcomata, only one was retroperitoneal, consequently any description of them must be founded on a collection of published cases, and that given by Steele being by far the most complete will be drawn upon largely. These growths may occur at any period of life, but are commonest under ten years of age and between forty and sixty; they are slightly more frequently in males than females; they begin about twice as often in the retroperitoneal lymph glands as in the retroperitoneal connective tissue. They spring most frequently from the centre of the abdominal retroperitoneal space, but may come from either side of it. They very rarely come from the pelvic retroperitoneal tissue. The tumour is usually lobulated and not larger than a man's head; it rarely invades surrounding structures; in its earliest stages it is hard and white, but it is very prone to degeneration, hence it often becomes cystic, and usually there is much hæmorrhage in it, so that the fluid withdrawn from the cyst is usually dark brown. Steele says that the microscopical examination of 44 cases gave the following results: spindle-celled sarcoma, 12; small round-celled sarcoma, 9; myxosarcoma, 8; spindle and round sarcoma, 7; lymphosarcoma, 5; large round-celled sarcoma, 2; and endothelioma, 1. Metastatic growths are frequently found, naturally most often in the liver.

The **symptoms** are at first indefinite; the patient complains most often that he does not feel well, that he has abdominal discomfort, amounting even to actual pain; he has indigestion, loss of appetite, and constipation or diarrhœa. But the symptoms which first direct attention to his trouble are most frequently œdema of the lower extremities, genitals, and abdominal wall: if the tumour press upon the one iliac vein rather than on the vena cava the œdema may be unilateral; and, secondly, severe pain along such nerves as may be compressed by the growth. As the tumour grows pain in the back becomes very marked, œdema increases, and pressure on the bowel leads to severe constipation or even actual obstruction. Sooner or later an abdominal tumour can be felt, its position varying with the region from which it sprang. It is mostly hard, often lobulated, and commonly, as it is frequently cystic, fluctuation or even a thrill can be obtained. The most important feature of the

tumour is that it is deep-seated, and as it grows forward either behind the ascending transverse or descending colon, it stretches apart the peritoneal attachment of the colon; this remains in front of the tumour, which therefore usually has a band of resonance over it. This resonance can often be demonstrated by inflating the colon. The stomach, too, may be in front of the tumour if it is high up. The small intestines are usually pushed aside, away from the point of origin of the tumour. The growth is nearly always immovable, as its base is wide and firm; rarely, if attached to the liver, it may ascend and descend a little with respiration. If it be low down in the abdomen it may be possible to feel it from the rectum or vagina. The disease is very rapid; the patient usually dies from exhaustion within a few months of the first symptoms.

The **diagnosis** from malignant disease of the suprarenals or kidney may be very difficult, but the separation of retroperitoneal sarcoma from non-malignant retroperitoneal tumour is usually easy, for in sarcomata the symptoms of pressure on veins and nerves and the rapid wasting are so striking. Retroperitoneal tuberculous glands can hardly give rise to any difficulty, for it is excessively rare for them to form a large mass that can be felt during life; they seldom cause great pain or œdema; there are nearly always signs of tubercle elsewhere, and sarcoma runs a much more rapid course.

Secondary sarcomatous deposits may take place in the retroperitoneal tissue, but they are generally overshadowed by the primary growth, which is commonly in the testicle or ovary.

Lipomata arising from retroperitoneal connective tissue are commoner than sarcomata, but are less important, for unless large they are not dangerous to life. They most frequently spring from the perirenal fat, especially on the right side, but they may come from any part of the back of the abdomen, and grow forward between the layers of peritoneum fixing the colon. They are not infrequent in the mesentery and great omentum; sometimes they appear to be enormous appendices epiploicæ, perhaps then occasionally they are detached and lie free in the abdominal cavity, and sometimes they grow out of the abdomen like an inguinal, femoral, or umbilical omental hernia, and then, inasmuch as they carry some peritoneum with them, they cause symptoms like those of an omental hernia, for which they are mistaken. They may be enormous, weighing over fifty pounds. Usually they are single, but frequently they are lobulated, and sometimes in such a way

as to suggest that one tumour resulted from the fusion of several. They are extremely liable to various changes; thus they may become myxomatous; often there is so much fibrous tissue that the tumour is really a fibro-lipoma; sometimes cartilage or bone is found in them, and they have been known to become sarcomatous. They may be diffuse or encapsuled.

Retroperitoneal lipomata are commoner in women than in men, and the sufferers are usually between thirty and fifty years old. The small lipomata may give rise to no symptoms, and the large, being soft and ill-defined, as felt through the abdominal wall, are not nearly so easy to distinguish as a sarcoma, and therefore they are often overlooked or mistaken for ascites; but with them, as with sarcomata, the bowel will be in front if they spring from the back of the abdomen. Inasmuch as they do not cause rapid wasting and intestinal obstruction, great pain or œdema, they are rarely confounded with retroperitoneal sarcomata, nevertheless it is important to be alive to their occurrence, for when detected early their removal is often easy and of great benefit to the patient, for if they are left to grow till they weigh many pounds, they render life a great burden and, further, they may at any time become sarcomatous.

It is important, when removing them during life, not, by so doing, to cut off the blood-supply to the colon. This has often been done, and has led to gangrene of the gut.

The last group of retroperitoneal tumours of sufficient medical interest to require notice are *cysts*. Some of these are sarcomata that have undergone cystic degeneration; others are ordinary hydatids, usually multiple when in the subperitoneal tissue, and they may be associated with hydatid of the liver; and others, such as dermoids of the broad ligament and presacral dermoids, are developed from fœtal relics. These from their position come most often under the care of the surgeon, but they may grow up from the pelvis so as to form large abdominal retroperitoneal tumours. But besides all these cysts there are others met with in the retroperitoneal tissue. Some are probably not true cysts, but are collections of altered blood or chyle shut in by adhesions and due to some antecedent rupture of a vessel. These may attain great size, and may spring from the subperitoneal tissue of the mesentery or omentum, but are as often, perhaps, derived from that at the back of the abdomen (see article, "Peritoneal Sanguineous Cysts"). The explanation of the others is not at all clear; they are thin-walled and contain clear fluid. Unless retroperitoneal cysts are so large as

to attract attention they produce very few signs. They rarely cause pain or œdema, and the sufferer usually at most complains of a vague sense of abdominal discomfort. The tumour is usually rounded and not lobulated like a sarcoma; it may be possible to tell by fluctuation or a thrill that it contains fluid, and if it come from the back of the abdomen its relation to the colon is the same as that already described for retroperitoneal sarcomata. These cysts produce no general symptoms unless they suppurate. Occasionally, but very rarely, they rupture. The treatment of them is surgical; removal is far better than aspiration, and it is well if possible to attack them without opening the peritoneal cavity.

Fibromata and myomata have been met with in the retroperitoneal tissue, but they are very rare. Traumatic inflammation of the tumour and recent effusion of blood, chyle, bile, fæces, urine and gas are more in the province of the surgeon than the physician. It has been known that bile fæces and gas have been effused as a result of ulceration.

In the above description it has been generally assumed that the retroperitoneal tumour sprang from the connective or lymphatic tissues under the peritoneum at the back of the abdomen, as indeed it nearly always does, but tumours growing between the layers of the mesentery or omentum are strictly retroperitoneal, although more often perhaps spoken of as subperitoneal. Tumours in either of these situations are even less common than those at the back of the abdomen. If the tumour be in the mesentery it is usually felt to be prominent in the neighbourhood of the umbilicus; it is often very mobile, especially in a transverse direction, and may even be rotated round a central axis; a piece of bowel lying on it may give rise to a resonant band across it, and the note around the tumour is resonant. Two-thirds of all mesenteric tumours are cystic, *e.g.* dermoid, chylous, malignant, sanguineous, serous, hydatid; one-third are solid. Of primary solid tumours lipomata are most frequent, fibromata, lymphomata, neuromata, angiomata, and lymphangiomata have been described; secondary carcinomata and sarcomata occur. Tumours of the omentum are nearly always part of a simple, malignant, or tubercular peritonitis (*q.v.*), and then form a solid tumour lying transversely across the abdomen, which has to be distinguished from tumours of the stomach or colon. Of other tumours of the mesentery lipomata are most common.

SUBPHRENIC ABSCESS

The term subphrenic abscess is applied to a localised collection of pus immediately below the diaphragm, either retroperitoneal or in the peritoneal cavity. It is localised by new-formed adhesions. The term is not usually applied to a localised collection of pus which is merely part of a chronic general peritonitis, *e.g.* such collections as might form as a part of tuberculous peritonitis.

A subphrenic abscess is never secondary to disease within the chest. By the time of the post-mortem suppuration in the pleural cavity or a gangrenous pneumonia often exists, and is frequently regarded as the cause of the subphrenic abscess, but the reverse is the truth, for a subphrenic abscess is always secondary to disease of an abdominal viscus. By far the most usual cause is ulcer of the stomach, and the common sequence is for adhesions to take place between the stomach and the left lobe of the liver or some part of the abdominal wall in its neighbourhood; perforation takes place into these adhesions, but they prevent general peritonitis, and a localised suppurative peritonitis ensues. As ulcers of the stomach are most frequent towards the pyloric end, the abscess usually forms just to the left of the falciform ligament, and is prevented by it from spreading towards the right, but it increases in size towards the left, and is commonly ultimately bounded to the right by the falciform ligament, above by the diaphragm, to the left by the vertical part of the diaphragm, the lower left ribs, and the spleen; below the abscess extends a little way over the front of the stomach, and it may be even to the colon, but usually on the front of the stomach the anterior abdominal wall is fixed to the stomach by adhesions, so that the anterior boundary of the abscess is the anterior abdominal wall, while the posterior is the anterior wall of the stomach, and above this the deep structures at the back of the abdomen, from which, however, the pus is separated by new-formed adhesions. The abscess here described would be unusually large, would push up the left half of the diaphragm, and would tend to extend upwards and towards the left, but all sizes, from those which are quite small to those occupying the above regions, exist. Very rarely a subphrenic abscess in the same way follows perforative disease of the pylorus, disease of the gall-bladder, of the duodenum, or of a long appendix which is directed upwards. In these cases the abscess is usually to the right of the falciform ligament.

Because a subphrenic abscess follows disease of a hollow viscus

it usually contains both air and pus. Often the original aperture in the viscus becomes closed by lymph, but even then the gas in the abscess may continue to increase from decomposition. If the abscess be large enough it presses on neighbouring structures, the most important effects being that the diaphragm and heart are pressed up and the liver is pressed down.

The inflammatory process almost always spreads through the diaphragm after the abscess has existed a short time, and pleurisy results. Pericarditis is not common. Sometimes the subphrenic abscess perforates the diaphragm, and then an empyema or a pyo-pneumothorax results, or if, owing to adhesive pleurisy, the perforation take place directly into the lung, this leads to gangrenous pneumonia.

The **symptoms** may begin with sudden pain in the upper part of the abdomen, and the patient may give a history which points to her having had gastric ulcer or other disease which leads to a subphrenic abscess. The pain increases and a swelling may form in the upper part of the abdomen; as this grows the ribs usually on the right become everted. The swelling is tender and dull all over unless gas be present in the abscess, when there will be resonance over the upper part of it; this resonant area may, if the diaphragm be pushed up, extend into the chest, and over it a *bruit d'airain* will be obtained, and neighbouring sounds such as intestinal gurgling, or the heart sounds may be very metallic. At first, during the acute stage, respiratory movement at the upper part of the abdomen and the lower part of the chest is absent or impaired. Later, the arch of the diaphragm being increased upwards, this muscle contracts at a greater mechanical advantage than normal, and the lung and the liver, if the subphrenic abscesses are right-sided, may descend more than usual during inspiration. If the abscess be large enough it hampers the breathing by pressure on the lung. There will be the usual symptoms of suppuration, viz. rigors, raised temperature, rapid pulse, wasting, anæmia, sweating, leucocytosis, and so forth. The existence of a subphrenic abscess may be suggested by the shadow thrown on the X-ray screen; and this may be useful not only for diagnosis, but as indicating the best situation for operation.

As disease of the chest so frequently follows, a patient suffering from subphrenic abscess usually shows evidence of thoracic complications. Thus a pleuritic rub or a pleuro-pericardial rub may often be heard; rarely signs of pericarditis may be present. If an empyema or a pyo-pneumothorax form they will show the usual physical signs, but it must be remembered that they are often of

very irregular shape owing to their limitation by adhesions; if the abscess have burst into the lung there will be a very foul purulent expectoration and the usual signs of gangrene of the lung.

The **diagnosis** of a subphrenic abscess is usually not difficult if the possibility of its existence be borne in mind. The most common mistake is to confound it with an empyema or pyo-pneumothorax. But if the collection of pus be primarily in the chest the upper level of the dulness is more or less horizontal; if below the diaphragm, it is arched upwards; the heart is displaced laterally when the fluid is in the chest, upwards when in the abdomen. The diaphragm is pressed up by an abdominal collection, and, as just explained, this leads to greater inspiratory movement; it is pressed down by a pleuritic collection, and this leads to lessened inspiratory movement. If the abscess has been punctured the pus naturally comes out more forcibly during inspiration if it be below the diaphragm, out more forcibly during expiration if it be above. Finally, it need hardly be added that the collection of pus occupies a lower position when below than when above the diaphragm. If the subphrenic collection be small, and the resulting supraphrenic collection be large, the exact diagnosis may be very difficult, but should it be found that air exists below and pus above, or that different collections of pus are by puncture obtained at different levels, it shows that the diaphragm is between the two collections, and that either there is no hole through it or that the hole has closed up. Sometimes it may be difficult to tell a subphrenic from a hepatic abscess. Occasionally, but very rarely, the abscess ruptures into the colon or stomach, and then the patient may recover. Rupture into the general peritoneal cavity is extremely uncommon.

The **treatment** is purely surgical and early diagnosis is very important, for it is very desirable to open the abscess before the supervention of an empyema or gangrenous pneumonia. The exact point at which the abscess is opened must depend upon its size and shape, but as a rule the most desirable spot is on the left side behind, in one of the lower intercostal spaces, for there free drainage is provided. Usually this does not lead to opening the pleural cavity, for this has become sealed at this spot by the secondary pleurisy, and even if the pleura be not adherent and the abscess cannot be reached below the pleura, it may be desirable, in order to obtain free drainage, to drain through the pleural cavity, and thus convert the case into one of pyo-pneumothorax.

W. Hale White.

TABLE SHOWING THE VASCULAR SUPPLY TO THE ABDOMEN

Branches of Abdominal Aorta.				Region Supplied.	Arterial Anastomosis.*	Venous Return.		Communications between the Portal and Systemic Circulations.
ABDOMINAL AORTA.			Œsophageal.	Lower end of œsophagus.	*Thoracic œsophageal and left phrenic.*	GASTRIC VEIN.		The œsophageal veins communicate— 1. With the œsophageal tributaries of the right and left azygos veins, which open into the superior vena cava. 2. With the diaphragmatic branches of the internal mammary veins, which open into the innominate, and so into the superior vena cava. 3. The œsophageal branches of the left phrenic, which opens into the inferior vena cava.
			Cardiac.	Cardiac end of stomach.				
		Gastric.	Gastric.	Front and back of stomach.	*Right and left gastro-epiploic.*			
			Great anterior gastric.	Front of cul de sac of stomach.	*Vasa brevia and left gastro-epiploic.*			
		Hepatic.		Left lobe of liver.	*Left hepatic.*	INFERIOR VENA CAVA.		
		Pancreatic.		Pancreas.	*Pancreatic of splenic and superior mesenteric.*	SPLENIC AND SUPERIOR MESENTERIC.		
		Superior pyloric.		Pylorus and lesser curvature of stomach.	*Gastric branch of coronary.*	PYLORIC.		
		Gastro-duodenal.	Right gastro-epiploic. Gastric. Omental.	Anterior and posterior surfaces of stomach. Omentum.	*Gastric, hepatic, and left gastro-epiploic. Omental of middle and left colic and of splenic.*	SUPERIOR MESENTERIC.		Rarely the gastro-epiploic veins communicate with the left renal, which opens into the inferior vena cava.
	Cœliac Axis.	Hepatic.	Superior pancreatico-duodenal.	Duodenum and pancreas.	*Inferior pancreatico-duodenal and superior mesenteric.*	" "		
			Inferior pyloric.	Pyloric end of stomach.	*Superior pyloric.*	PYLORIC.		
			Right terminal. Cystic.	Right lobe of liver. Gall-bladder.		INFERIOR VENA CAVA. PORTAL VEIN.		The portal vein communicates— 1. By means of the accessory portal veins of Sappey (in the connective tissue around the liver) with tributaries of the phrenics which open into the inferior vena cava, and with tributaries of the internal mammary and right azygos which open into the superior vena cava.
			Left terminal.	Spigelian and left lobe of liver.		INFERIOR VENA CAVA.		
		Splenic.	Smaller pancreatic.	Pancreas.	*Pancreatic of hepatic and superior mesenteric.*			2. By means of the para-umbilical vein (a constant vessel passing from the left division of the portal vein along the round ligament to the umbilicus), with the deep epigastric and so with the external iliac and the inferior vena cava, and with the superior epigastric and so with the internal mammary and the superior vena cava. By the communications between the deep and superficial epigastric veins the portal blood may enter the superficial epigastric and superficial circumflex iliac veins, and thence gain the saphenous vein and the inferior vena cava; and by the lower intercostals, musculo-phrenic, sternal, and the thoracic branches of the axillary vein open into the superior vena cava.
			Large pancreatic.	Pancreas and pancreatic duct.	" "			
			Left gastro-epiploic. Gastric. Omental.	Front and back of stomach. Omentum.	*Right gastro-epiploic, gastric and hepatic. Omental of middle and left colic and of hepatic.*	SPLENIC.		
			Vasa brevia.	Greater cul de sac of stomach.	*Gastric and left gastro-epiploic.*			3. By means of the sub-peritoneal plexus wherein duodenal, colic, rectal, and pancreatic branches anastomose with branches of the phrenic, lumbar, ilio-lumbar, circumflex iliac, lower intercostal, epigastric, middle and lateral sacral, with the superior and inferior vena cava.
			Terminal.	Spleen.				
	Superior Mesenteric.		Inferior pancreatico-duodenal.	Pancreas and duodenum.	*Superior pancreatico-duodenal.*			4. Rarely, by means of branches from the descending colon anastomosing with branches of the left spermatic, with the left renal, and so with the inferior vena cava.
			Middle colic.	Transverse colon.	*Right and left colic.*			
			Right colic.	Ascending colon.	*Ileo-colic and middle colic.*	SUPERIOR MESENTERIC.	PORTAL, which also receives the PYLORIC, GASTRIC, and CYSTIC.	5. Rarely, by an anastomosis of the vasa brevia with branches of the left phrenic, with the inferior vena cava.
			Ileo-colic. Colic. Ileac.	Termination of ileum, cæcum, ascending colon, and vermiform appendix.	*Right colic. Superior mesenteric.*			6. By means of the hæmorrhoidal plexus, with the middle hæmorrhoidal of the inferior vesical and the inferior hæmorrhoidal of the internal pudic, and so with the internal iliac and the inferior vena cava.
			Intestinal.	Jejunum, ileum, mesentery, and mesenteric glands.	*Ileo-colic.*			
	Inferior Mesenteric.		Left colic.	Transverse colon and descending colon.	*Middle colic and sigmoid.*			
			Sigmoid.	Sigmoid.	*Left colic and superior hæmorrhoidal.*	INFERIOR MESENTERIC.		
			Superior hæmorrhoidal.	Rectum.	*Sigmoid and middle and inferior hæmorrhoidal from internal iliac.*			

* *There is a sub-peritoneal arterial plexus wherein the following visceral arteries: hepatic, renal, suprarenal, pancreatic, duodenal, cæcal, colic, rectal, and spermatic anastomose with the following parietal vessels: phrenic, lumbar, ilio-lumbar, circumflex iliac, lower intercostal, epigastric, middle sacral, lateral sacral, superficial pudic and perinæal.*

(Face page 52).

DISEASES OF THE ABDOMINAL BLOOD-VESSELS

EXCESSIVE PULSATION OF THE ABDOMINAL AORTA

This is, as far as is known, an entirely functional disorder of the aorta. It occurs between the ages of twenty-five and forty-five, and more frequently in women than in men. Its recognition is of the greatest importance on account of its liability to be mistaken for aneurysm. Its **etiology** is obscure. It has been traced to strains; it may be associated with accumulation of fæces in the transverse colon and other gastro-intestinal disturbances, with atheroma of the aorta, and with displacement of the vessel by enlarged glands or tumours. It has been known to follow syphilis and to be associated with gout. Patients affected with this disorder are usually thin, anæmic, extremely nervous, hysterical, and decidedly hypochondriacal. It is a condition which is found to be not infrequently associated with movable kidney and enteroptosis.

The **symptoms** are: pain in the abdomen, a feeling of discomfort and distress, a sensation of pulsation and throbbing over the abdominal aorta, nausea, retching, sickness, constipation, and, in some cases, melæna and hæmatemesis. The pain is usually most marked in the epigastric and umbilical regions, and is occasionally felt in the back; it may be paroxysmal in character, is worse when the throbbing is marked and violent, and is generally most troublesome at night. The pain may cause insomnia, restlessness, and a feeling of despondency. The sensation of throbbing may cause considerable discomfort and great alarm. Hæmorrhages from the stomach and intestine are usually small but repeated, and, as pointed out by Dr. Sidney Phillips (*Brit. Med. Journ.* 8th Oct. 1887), occur with considerable frequency with no evidence or indication of ulceration as an explanation. Dryness of the mouth and throat may also be a prominent symptom.

A careful physical examination reveals a flat abdomen with thin flaccid walls and marked pulsation in the epigastrium. On palpation the pulsation may be felt to be forcible, abrupt, and jerking in character, but the normal cylindrical outline of the aorta may be determined to be quite free from any saccular bulging or fusiform dilatation. No thrill is felt, as a rule, over the

pulsation. On lightly applying a stethoscope to the pulsating area of the abdominal wall there will be no audible murmur, but firm pressure, sufficient to slightly compress the aorta, will bring out a systolic murmur. The kidneys may be felt to be movable, and when the patient assumes the erect posture the lower part of the abdomen may become prominent and the epigastrium flatter. The heart and lungs are generally normal, and there is no evidence of any disease of the other viscera. In some cases an abdominal tumour may be felt which is elastic to the touch, tympanitic on percussion, and due probably to dilatation of the stomach or to distension of a part of the intestine with gas.

With the exception of slight atheroma, post-mortem examinations have revealed no changes to clear up the pathology of pulsating abdominal aorta. It is possibly the result of a disturbance of the vaso-motor nerves or solar plexuses, causing a paralysis of the vessel wall, reflexly induced by irritative lesions in the alimentary tract.

The pain is probably the result of the stretching of the nerve plexuses caused by the excessive pulsation. The hæmorrhages may be due to the rupture of small vessels brought about by the excessive pulsation, or to vaso-motor paralysis allowing undue engorgement followed by rupture from over-distension. The tumour caused by local distension of the intestine may be from paralysis of that part of the gut.

The **diagnosis** from abdominal aneurysm must be made by determining the absence of a tumour connected with the vessel and of marked expansile pulsation. If the wall of the abdomen be rigid, and there is any doubt as to the existence of an aneurysm, the patient should be examined under an anæsthetic, when the outline of the abdominal aorta can be determined and a definite conclusion arrived at. The extreme rarity of the occurrence of aneurysm of this part of the aorta should also be borne in mind, and the physician should hesitate to diagnose such a rare and serious disease unless the cardinal signs of aneurysm—pain, tumour, expansile pulsation, a murmur audible without exciting any pressure with the stethoscope and pressure signs—are present.

Prognosis.—The affection is rarely if ever fatal. The condition may pass off, but is liable to recur. Many cases are, unfortunately, very persistent, troublesome, and intractable, and may determine a condition of chronic invalidism.

Treatment. — The patient should be put under the best possible hygienic conditions, and should have exercise and plenty

of fresh air. The diet should be plain and simple, and stimulants should be avoided. Patients must be encouraged not to discuss or brood over their troubles. The bowels should be carefully regulated. Full doses of bromide of potassium, valerianate of zinc and small doses of tincture of aconite are useful in some cases, especially those with hysterical manifestations, and nitro-glycerine has been of benefit in others. If the pain be severe the judicious administration of opium or morphia may be necessary. If there be a previous history of syphilis potassium iodide should be administered, and relief has often followed. Movable kidney and enteroptosis should be treated by the application of a suitable belt or bandage, carefully adjusted to the abdomen whilst the patient is lying down, in order to support and strengthen the abdominal wall.

ANEURYSM OF THE ABDOMINAL AORTA

That this disease is of rare occurrence is well demonstrated by an analysis of the post-mortem examinations which have been performed at Guy's Hospital between the years 1854-1900 inclusive. During this period of forty-seven years 18,678 necropsies were made, and of these 325 were cases of aneurysm of the aorta, of which 54 were of the abdominal portion. It has been stated that, of all cases of aneurysm of the aorta, the abdominal part is only affected in 5 per cent; the above figures, however, indicate a somewhat greater frequency, viz. 16 per cent.

Etiology.—Males are much more likely to be affected than females, for of the 54 cases 49 were men, which gives a percentage of 91, or about 10 to 1.

The age incidence is as follows: 2 under 20, 11 between 21 and 30, 23 between 31 and 40, 8 between 41 and 50, 10 above 50; thus 34 or 63 per cent occurred between the ages of 21 and 40, and 77 per cent between 21 and 50. This analysis does not bear out the statement that it is a disease of middle age, when of these 54 consecutive cases 36, or 63 per cent, occurred in the third decade. These figures correspond to the analysis of cases collected by Crisp, of whose 59 cases 33 were under 40 years of age.

The two main factors in its causation are without doubt strains and syphilis, and in many cases these two causes are associated together, for the artery weakened, often locally, as a result of this disease, is more likely to yield to an increase of pressure resulting from a sudden strain or violent muscular exertion. The fact that

it is so much more prevalent in men points to the probability of some marked difference of environment as compared with women, and this difference is at once evident when their respective occupations are compared. It is commoner in the labouring classes than the well-to-do, for men of the former class earn their living by engaging in hard manual labour, often subjecting themselves to violent muscular efforts. It may follow injuries to the back or abdomen, heavy lifting, a fall from a height, or a sudden wrench.

An analysis of the previous history of patients shows that in a large number of cases they have suffered from attacks of acute rheumatism, or from chronic alcoholism, but whether the changes produced by these conditions have any influence in causing the disease is not evident.

Morbid anatomy and pathology.—There are five varieties of aneurysm of the abdominal aorta, viz. saccular, diffuse, dissecting, fusiform, and arterio-venous. Of these the saccular and diffuse are by far the commonest, and the arterio-venous the rarest, there being only 2 cases of this last-mentioned kind out of 54. The diffuse form is usually the result of rupture of a saccular aneurysm into the adjacent tissues. The leakage of blood may be quite gradual but slowly progressive, until a large tumour is produced, causing displacement of and pressure on adjacent structures. On the other hand, a copious and sudden outpouring of blood may produce during life all the signs and symptoms of a large intestinal hæmorrhage, which may rapidly prove fatal; or, should recovery take place, the patient remains blanched and a large abdominal tumour is found to have suddenly arisen, which may be observed to gradually increase in size as further effusion occurs.

The saccular and diffuse forms may start from any part of the vessel, but most frequently spring from the region of the orifice of the cœliac axis, an analysis of the 54 cases showing 36 (67 per cent) in this position, 13 above, 12 below, and 11 at the orifice. Five occurred at the origin of the superior mesenteric, and 3 at the origin of one of the renal arteries. All of these were either saccular or diffuse. The fusiform aneurysms involve the lower part of the vessel; the dissecting, the whole of the abdominal aorta, and often a part or the whole of the thoracic aorta as well; the arterio-venous the lowest part at or just above the bifurcation. Aneurysms may arise from the anterior, lateral, or posterior aspects of the artery. In size they vary from a slight local bulging to a tumour several inches in diameter.

One of the chief effects of the increase in size of the sac is erosion of the vertebræ, a condition which is more likely to occur when it arises from the posterior or lateral wall of the vessel. The amount of destruction may vary from a slight superficial erosion to an almost complete destruction of a body or bodies of the lowest dorsal and upper lumbar vertebræ, leading to pressure on the spinal cord and nerve roots. A curious and constant feature of this destruction of bone is the invariability with which the intervertebral cartilaginous discs escape destruction, a striking contrast to the effect of spinal caries from other causes, in which the discs are destroyed first and often more extensively than the bone. The proportion of cases in which erosion occurs is between 40 and 50 per cent, depending entirely on the direction in which the aneurysm enlarges.

When death occurs as a direct result of the aneurysm rupture of the sac is almost always found as the cause. Of the 54 cases, rupture into the retroperitoneal tissues occurred in 19, the peritoneal cavity 12, pleural cavity 8, the mediastinum 2, pericardial sac 1, and the mesentery 1. In addition, rupture may occur into the stomach, duodenum, lung, pelvis of the kidney, bladder, gall-bladder, or bile duct. When the rupture takes place into the peritoneal, pleural, or pericardial cavities an enormous quantity of blood and blood clot is found, and in the case of the pleural cavity the corresponding lung is compressed and airless, and the heart is displaced, being pushed over to the opposite side of the thorax. When the rupture occurs into the retro-peritoneal tissues the effused blood pushes forward the peritoneum, and the structures lying in front of it become displaced; the kidney on the affected side may be pushed forwards or downwards; the ureter may be kinked or obstructed, with hydronephrosis as a result; the duodenum may be compressed or twisted, producing dilatation of the stomach; and the pancreas and other viscera may also have their normal positions altered. The blood may plough up the substance of the quadratus lumborum and psoas muscles, and burrow downwards into the thigh and backwards into the loin. In one case the genito-crural nerve was dissected out by the blood clot. The blood may extravasate behind the diaphragm into the posterior mediastinum and compress and obstruct the œsophagus, and may even extend upwards and cause a swelling in the neck. The inferior vena cava may be displaced or pressed on by the sac of the aneurysm, but it is by no means a common result. There may be a communication between the inferior vena cava and the

aorta, but this only occurred in two cases, in both of which the aneurysm involved the aorta at its bifurcation.

Œdema of the legs and lower part of the body may result from obstruction of the inferior vena cava, and this condition is particularly well marked, and is associated with enormous distension of the superficial veins of the leg, scrotum, and lower part of the abdomen if there be an anterio-venous aneurysm. Occasionally the renal veins are pressed on and thrombosed, and the kidney is congested and ecchymosed. In one case blood was effused into the right spermatic cord and the corresponding half of the right scrotum was much ecchymosed. The spermatic artery may be obstructed and atrophy of the testicle be found as a result. If the aneurysm arise from the lower part of the aorta one of the iliac veins may be compressed, producing œdema of the corresponding leg. An aneurysm arising from the right side of the upper part of the aorta in the region of the cœliac axis may press on the bile ducts or portal vein and give rise to jaundice or ascites.

In more than half the cases it is customary to find ante-mortem thrombus in the aneurysm, which may vary from a thin layer to a mass completely filling the sac; this latter result, which is aimed at by the various methods of treatment, is however unfortunately extremely rare. As one or more of the branches of the abdominal aorta are generally found to arise from the sac of the aneurysm, the effect of the thrombus is in some cases to completely block such a vessel. Or the thrombus may increase in size and finally block the lower end of the aorta, or pieces of the thrombus may become loose and embolic blocking of the renal, superior mesenteric, inferior mesenteric, or iliac vessels result. Atrophy of the kidney may follow embolism of the renal artery; infarction and sloughing of the intestine may result from embolism of the mesenteric arteries, and gangrene of the legs from embolism of the iliac arteries or blocking of the lower end of the aorta.

In most cases the aorta is atheromatous throughout, but, on the other hand, with the exception of the part involved by the aneurysm, the vessel may be perfectly healthy, a condition which is noticed in young patients and in those who have never suffered from syphilis, and who have not been addicted to alcoholism. When an aneurysm arises from an apparently healthy aorta it is usually the result of an injury to the inner walls of the vessels from a severe strain or violent blow.

Peritonitis is a very rare complication, and is most likely to occur if pressure be adopted as the method of treatment.

Symptoms.—*Pain* is by far the most constant and is usually the earliest indication of aneurysm of the abdominal aorta. It is quite independent of the situation of the sac, and varies considerably in its position, character, severity, and duration. It may be constant and uniform, or intense and paroxysmal. It is usually complained of in the back or abdomen, especially in the epigastrium; it may be situated in the loin, groin, hip, sacro-iliac region, or chest. It may shoot round the body, down into the legs, or into the testicles. Its character differs, being variously described as severe, continuous, dull, heavy, aching, throbbing, gnawing, boring, tearing, or neuralgic. A continuous dull aching pain may suddenly become intense and paroxysmal in character. Although pain is usually associated with erosion of the vertebræ, it does not necessarily follow that this pathological change is always the cause of it, for in some cases, in spite of extensive bone destruction, the pain is slight. On the other hand, the pain may be very severe and paroxysmal in character without the slightest erosion of the bodies of the vertebræ, and it is then probably due to stretching of the nerve filaments over the sac. If the erosion be extensive the nerve roots may be pressed on, and the pain is then intense and corresponds to their distribution. Diffuse aneurysms may press on various nerves, such as the last dorsal when the pain is felt over the crest of the ileum, the genito-crural nerve with pain shooting down into the testicle, the sciatic nerve or the anterior crural with pain down the back or front of the leg. The pain due to erosion of the vertebræ is usually aggravated by pressure or by extending or compressing the spine. If the ureter be pushed forwards and put on the stretch by the aneurysm, severe attacks of pain, commencing in the loin and radiating in the direction of the ureter to the testicle, penis, and upper part of the thigh, are produced, and are not uncommonly mistaken for attacks of renal colic due to the presence of calculi. The testicle under these circumstances may become swollen and tender.

Pain, whatever its character may be, is generally increased by movements of the body, by walking, and by certain attitudes which vary according to the position of the aneurysm. Many patients cannot lie on the left side; others cannot keep still, and have to be constantly shifting their position; others cannot remain flat on their back, and some cannot sit up. As a general rule the easiest position is sitting up in bed propped up with pillows and with the body slightly bent forwards. Insomnia is a common and troublesome symptom, and is a result of the severe pain; it occurs in

more than a third of the cases. Gastric symptoms are often prominent, such as retching, vomiting, and flatulence, and may be due to the gastric branches of the solar plexus being involved, or to actual pressure on the duodenum or stomach. The symptoms of dilated stomach may be present when the duodenum or pylorus is compressed. The cardiac end of the stomach or lower end of the œsophagus may be pressed on by an aneurysm arising from the upper part of the abdominal aorta, or from rupture into the posterior mediastinum; well-marked dysphagia may result and lead to a diagnosis of œsophageal obstruction from malignant stricture. If the aneurysm rupture into the stomach or duodenum there will be profuse hæmatemesis and melæna. Constipation was stated to have been present in 16 out of the 54 cases. Jaundice and ascites may occur if the bile duct and portal vein be pressed on and obstructed. Dyspnœa is a common symptom, and may be caused by the extension of the aneurysm upwards, pushing up the diaphragm and compressing the lungs. Sudden and urgent dyspnœa is produced when an aneurysm ruptures into either of the pleural cavities. Cough is another symptom. Sometimes the earliest symptom complained of is a sense of discomfort, throbbing, and pulsation in the epigastrium, and occasionally the patient is conscious of the presence of a pulsating tumour quite early in the disease, and it may be for this that the medical man is consulted.

Loss of power and sensation in the legs, a zone of hyperæsthesia around the upper part of the abdomen, and involuntary passage of urine and fæces, may occur as a result of extensive erosion of the bodies of the vertebræ with consequent pressure on the spinal cord. Somewhat similar symptoms may be produced if the aorta become obstructed with a large embolus or by the extension of a thrombus from the aneurysmal sac, conditions which lead to anæmia of the spinal cord and to a sudden cutting off of the blood-supply to the muscles and nerves of the legs and lower part of the body. Embolism of any of the branches of the abdominal aorta may occur, but it is a rare complication. Embolism of the gastric arteries may cause hæmatemesis; of the splenic artery, pain and tenderness over the spleen; of the renal arteries, pain in the loins, hæmaturia, and albuminuria; of the superior and inferior mesenteric arteries, sudden severe abdominal pain, distension of the abdomen, vomiting, hæmorrhage from the rectum and collapse, followed by death in a few days. If one of the iliac arteries become obstructed there is a feeling of pins and needles in the foot and leg, numbness and coldness, followed finally by gangrene. Emaciation, increasing

weakness, and prostration occur as a result of the constant wearing pain and suffering, and the patient dies from exhaustion and syncope.

In quite half the cases death is sudden from rupture of the sac into the peritoneal or pleural cavities. If the rupture occur into the retroperitoneal tissues the escape of blood is much slower, and the patient may at first experience relief from the intensity of the pain, but he becomes increasingly anæmic and gradually dies from exhaustion, or succumbs to another and more extensive hæmorrhage. The symptoms of rupture are sudden collapse, intense pain, restlessness, pallor, profuse clammy perspiration, coldness of the extremities, a rapid thready pulse, and a sudden subsidence of the tumour if the rupture occur into the pleural or peritoneal cavity.

Physical signs.—The chief indications of this disease to be obtained by a physical examination are: the presence of a tumour with expansile pulsation, occasionally a thrill, and more frequently a systolic murmur audible over its site. In addition to these, there are the signs produced as a result of pressure on the various intra-abdominal structures, and the extravasation of the blood into the retroperitoneal tissues.

The position of the tumour varies, and is dependent in the case of saccular aneurysm on the part of the aorta from which it arises, and in the case of diffuse aneurysm on the direction in which the blood extravasates after retroperitoneal rupture has occurred.

The tumour may be sufficiently large to cause a local bulging or prominence of the abdominal parietes, and the part of the abdomen most frequently affected is the epigastrium; this occurs when the sac arises from the aorta in the region of the cœliac axis and projects anteriorly. If the sac increase in size to the left the bulging may be noticed in the left hypochondrium or left flank, and it may even cause a marked projection of the lower ribs. When the sac enlarges posteriorly a tumour may be noticed in the lumbar region and back. If it extend towards the right the tumour will be in the right hypochondriac region or right loin. If the aneurysm be high up and just below the diaphragm no definite bulging is as a rule apparent. When the sac arises from the aorta much below the cœliac axis the tumour is in the umbilical region. In cases of diffuse aneurysm, when the blood has burrowed into the tissue of the quadratus and psoas muscles, the tumour may resemble a renal enlargement, and if the effusion extend below Poupart's ligament in the sheath of the psoas a tumour may become obvious in the

groin and below it, which may simulate psoas abscess. In some cases the tumour may be in the left iliac region.

Inspection over the prominent area will show a pulsation which may be forcible and very marked, or slight and almost imperceptible. This is most marked in a saccular aneurysm which contains little or no thrombus; if the sac be filled with adherent clot, or if the aneurysm be diffuse, the pulsation may be slight. Pulsation may also be a noticeable feature even if the aneurysm be not sufficiently large to cause a bulging of the parietes.

On palpation a tumour may be felt and the pulsation determined to be distinctly expansile. If the patient be placed in the knee-elbow position the pulsation does not disappear as it does if it be due to a tumour lying over the front of a healthy aorta. Dr. Osler states that sometimes the pulsation is double when the sac is large and in contact with the pericardium. A saccular aneurysm feels globular, smooth, regular, and elastic, and it may be compressible, tender, and even painful. If it be filled with adherent organised thrombus it is hard, firm, non-compressible, and non-expansile, and if the opening into the aorta be blocked there is little or no pulsation. A diffuse aneurysm giving rise to a tumour in the loin may simulate a renal enlargement, and pulsation may be present or entirely absent. In some cases the kidney can be felt lying in front of the tumour. An abdominal tumour due to aneurysm of the abdominal aorta is not as a rule influenced by the respiratory movements, nor is it at all movable unless it arises at the orifice of one of the large branches, and the sac is formed partly by a distension of the aorta and partly by a dilatation of the particular branch itself. In some cases a pulsating tumour may be detected in the position in which a psoas abscess is usually felt. The pulsation in the femoral arteries may be unequal. Pressure on the tumour may cause a distinct increase of tension in these arteries, and if the pressure be suddenly relaxed, a fall of tension and intermittency of the femoral pulse may be noticed for a few seconds.

A thrill can occasionally be felt on palpating the tumour, and its rhythm is usually systolic or slightly post-systolic. There may be evidence of displacement of the heart upwards or of the liver downwards.

If the tumour be large it may yield a dull note on percussion, which may be continuous with the liver dulness. If the sac of the aneurysm be small or moderate in size, the stomach, colon, or intestines may be lying in front of it according to its position, and

on percussion the note is resonant or subresonant. If the tumour project posteriorly or in the loins the note is dull over the prominent area.

A murmur is audible over the tumour in about 50 per cent of the cases. It may be heard in front or behind, but it is usually best marked over the most prominent part of the aneurysm. It varies in intensity and character, being in one case very loud and in another very faint; it may be soft or blowing or musical; it is usually systolic, but may be post-systolic. It may be altered by a change of posture or by pressure, but is audible when the stethoscope is applied quite lightly to the abdominal wall. Dr. Osler states that a diastolic murmur is occasionally present, very soft in quality. The murmur may be absent if the aneurysm is diffuse, or if a saccular aneurysm be filled with adherent clot. In a case of arterio-venous aneurysm involving the aorta just above the bifurcation and the lower part of the inferior vena cava and the right iliac vein, a loud systolic bruit was heard along the course of the aorta, all over the abdomen, and down the outer side and the back of the thigh.

If the aneurysm press on the cardiac end of the stomach or lower end of the œsophagus the patient rapidly emaciates, and the abdomen is flat and retracted. If the duodenum or pylorus be obstructed by pressure or kinked by displacement the stomach becomes dilated, and the physical signs of this condition are present. If the aneurysm erode the vertebræ and press on the spinal cord there is weakness of the lower extremities, loss of sensation, a wide band of hyperæsthesia around the body at the seat of the lesion, exaggerated knee-jerks, extensor plantar response, and involuntary passage of urine and fæces, according to the part of the cord involved. When blood has extravasated from an aneurysm into the retroperitoneal tissues, and pushed forward the kidney and involved the ureter, the testicle on the same side may become swollen and tender.

If the aneurysm rupture into the peritoneal or pleural cavities the tumour and the pulsation may disappear, and in the latter case the signs of a large pleural effusion may suddenly become apparent.

Diagnosis.—The presence in the abdomen of a tumour with distinct expansile pulsation and a systolic bruit, associated with severe pain, points to the presence of an aneurysm of the abdominal aorta or of one of its branches. In a large proportion of the cases all the cardinal signs are not present, and then the diagnosis

presents a good deal of difficulty, and is often not made until rupture takes place, or even until a post-mortem examination is performed. An analysis of the 54 cases from the Guy's Hospital records shows: the presence of an abdominal tumour in 31, of pulsation in 35, of expansile pulsation in 8 only, and of a systolic bruit in 26. Out of the 54 cases a correct diagnosis appears to have been made in 18 only. Incorrect diagnoses of renal calculus, malignant tumours lying in front of the aorta, spinal caries, lead colic, sarcoma of the kidney, nephritis, perinephritis, lumbago, pneumothorax, pleuritic effusion, epithelioma of the œsophagus, and malingering were made. A diagnosis of abdominal aneurysm is much more frequently made when it is not present than an incorrect diagnosis when it is present. The condition most frequently mistaken for it is "pulsating abdominal aorta," but the fact that this is much more common in women than in men should make the physician very chary of committing himself to a diagnosis of abdominal aneurysm in a female; for, as above stated, of the 54 cases there were only 5 females affected, and of these 1 was a commencing aneurysm of the end of the aorta, the result of embolism in a child who was suffering from infective endocarditis, so that there were really only 4 cases of abdominal aneurysm in women. The essential features of undue aortic pulsation have been previously recounted (see p. 523).

Marked epigastric pulsation from an enlarged right ventricle and a pulsating liver might be mistaken for abdominal aneurysm, but should present no difficulty, for it is extremely rare to find any hypertrophy or dilatation of the heart associated with abdominal, or even thoracic aneurysm, unless the dilatation have involved the intrapericardial portion and caused aortic regurgitation.

Malignant tumours lying over the aorta, especially of the pylorus, often pulsate on account of their close relationship to the aorta. The pulsation, however, is not expansile, and the tumour may be distinctly movable on respiration, which is a most unusual characteristic of aneurysm of the abdominal aorta. Another distinguishing test is to place the patient in the knee-elbow position, when the pulsation will disappear if it be due to a malignant tumour, but will not do so if the tumour be an aneurysm.

Renal calculi and the pathological changes associated with it are often most difficult to distinguish from aneurysm, for when the latter pushes forward the kidney and stretches the ureter the pain may be severe and paroxysmal in character, may shoot along the course of the ureter into the scrotum, and the testicle may become

swollen, tender, painful, and retracted. In cases presenting such a group of renal symptoms a careful estimate of the nature of the patient's occupation, of a previous history of syphilis, of sudden strain or injury, followed by pain in the abdomen or back, and the presence of any pulsation or bruit, should indicate the possible presence of a diffuse aneurysm of the abdominal aorta.

An examination by the X-rays should be made in all doubtful cases, and the points to examine for are: a large pulsating area connected with the aorta, and a loss of the normal contour of the bodies of the vertebræ from erosion. If renal calculi were present they might be seen on the fluorescent screen, or would be demonstrated, in a very large proportion of the cases, by a photograph.

If the aneurysm have eroded the vertebræ and caused paraplegia from compression, the diagnosis must be made from spinal caries, from a growth pressing on the cord, or from myelitis and from paralysis due to obstruction of the aorta by a thrombus. A careful examination must be made for the presence of a deep-seated abdominal tumour, or one presenting at the back, and for expansile pulsation or a bruit. If the paralysis be due to blocking of the lower end of the aorta there would be no pulsation in the femoral arteries.

The diagnosis of rupture of an abdominal aneurysm must be made from the conditions causing sudden pain, collapse, and anæmia, *e.g.* perforative peritonitis, ruptured extra-uterine fœtation, acute intestinal obstruction, and acute hæmorrhagic pancreatitis.

The **prognosis** is very bad. There were only 2 cases of cured aneurysm of the abdominal aorta found in 18,678 necropsies. One died from cerebral hæmorrhage, the other from aortic incompetence. The average duration of life from the time the aneurysm became manifest was about 13 months. The shortest duration was a week and the longest 12 years.

Death is usually sudden from rupture into the peritoneal, pleural, or pericardial cavities, hollow viscera, or lungs, but when the rupture is into the retroperitoneal tissues death is, as a rule, much slower. If the aneurysm press on the œsophagus or cardiac end of the stomach death takes place from exhaustion and starvation. If the renal arteries be obstructed death results from uræmia. Death may occur without rupture having taken place, and be due to exhaustion and cardiac failure, or it might be quite independent of the aneurysm, and be the result of some intercurrent disease.

Treatment.—The general, dietetic, and medicinal treatment

is the same as for aneurysm of the thoracic aorta, and has been fully considered under that heading (Vol. IV. p. 456). The drugs which are usually administered are acetate of lead, potassium iodide in doses of from 10 to 30 grains, aconite, ergotine, and calcium chloride. Morphia is generally necessary to allay the pain. Successful cases of cure brought about by pressure have been recorded, but they are few and far between. The pressure may be proximal or distal. If the aneurysm arise from the lower half of the abdominal aorta proximal pressure may be adopted, but as in the greater number of cases the neighbourhood of the cœliac axis is usually involved, distal pressure is the only method of applying this treatment. The object of the treatment is to promote the formation of adherent thrombi in the sac. The bowels should be well opened and an anæsthetic administered. The pressure may be applied by means of Lister's tourniquet. Great care must be exercised on account of the possibility of inducing peritonitis or enteritis, and care must be taken to avoid pressing on the renal arteries, as suppression of urine may follow.

Galvano puncture, the introduction of wire into the sac, and M'Ewen's method of producing white thrombi, are all unsatisfactory methods of treatment which hold out very little hope of cure or even of amelioration.

Subcutaneous injections into the gluteal regions of a sterilised 2 per cent solution of gelatin was first introduced by Lanceraux for the treatment of aneurysm, with a view to the production of clotting in the sac, but the results have not been promising. Great care must be exercised in the preparation of the gelatin to render it absolutely sterile, as tetanus has followed the injection of this substance.

Aneurysms of the Branches of the Abdominal Aorta

Aneurysms of the other arteries of the abdomen are even of rarer occurrence than of the abdominal aorta. At Guy's Hospital there were 6 cases of aneurysm of the cœliac axis, 4 of the splenic artery, 1 of the gastro-epiploica, coronary artery of the stomach, and of the cystic artery, 4 of the superior mesenteric, and 1 of the renal, between the years 1854 and 1890 inclusive, during which time 18,678 necropsies had been performed.

According to Lebert (cited by Quincke in von Ziemssen's *Cyclopædia*), out of 39 cases of aneurysm of the abdominal arteries, excluding the aortic trunk, the superior mesenteric and splenic

were affected 10 times, the hepatic and its branches 8 times, the cœliac and inferior mesenteric 3 times, and the renal twice; in 3 instances the aneurysms were multiple, in 36 they were solitary.

ANEURYSM OF THE CŒLIAC AXIS

It is often difficult to say, even at a post-mortem examination, after a careful dissection, whether an aneurysm in this position is entirely due to a dilatation of this vessel or of the abdominal aorta at the origin of the cœliac axis. If the orifice of this artery be found to be much dilated the aneurysm ought not to be considered as one of the cœliac axis alone, but must be classified as one of the abdominal aorta involving the cœliac axis.

The symptoms, physical signs, and results of aneurysm of this vessel are much the same as those produced by aneurysm of the aorta in this region.

The tumour projects anteriorly from the aorta and may be distinctly movable from above downwards, and from side to side. It may also move downwards when the patient takes a deep breath. It may press on the bile ducts and give rise to jaundice. It may cause hæmatemesis and melæna. One patient after a fall on the back across a barge suffered from abdominal pain, wasting, and vomiting, and had been treated for dyspepsia for several weeks. One day, when taking a walk, he fell down and died soon afterwards. It was found at the post-mortem examination that he had a large saccular aneurysm of the cœliac axis which had ruptured into the peritoneal cavity. Another case was mistaken for dilated stomach and gastric ulcer, the aneurysm having compressed the duodenum. In another case, on account of the severe pain being relieved by pressure, a diagnosis of lead colic was suggested. Death is usually very sudden from rupture into the peritoneal cavity.

ANEURYSM OF THE HEPATIC ARTERY

This is very rare. The case recorded in the Guy's Hospital post-mortem reports was a student, æt. eighteen, who was admitted for an attack of pneumonia, which terminated by lysis on the eighth, ninth, and tenth days of the illness. For three days after, the temperature was normal. On the fourteenth day a hectic temperature commenced and continued until his death. On the twentieth day of his illness his abdomen became dis-

tended and jaundice developed. On the twenty-fourth a localised empyema under the right nipple was found and evacuated. A fulness had been noticed over the left lobe of his liver, but no pulsation. He died quite suddenly a month after the commencement of his illness. At the post-mortem examination a large quantity of blood clot and blood-stained serum was found in the peritoneal cavity. There was an aneurysm of the right branch of the hepatic artery, about the size of a Tangerine orange, which had ruptured into the peritoneal cavity. It had pressed on the bile duct and the portal vein. There was another aneurysm about the size of a walnut springing from the left branch of the hepatic artery, about half an inch within the liver substance. Adherent ante-mortem clot was found in both sacs. The aneurysm in this case was probably the result of septic embolism. Embolic plugging, septic or non-septic, is probably the most frequent cause, and this applies to most of the abdominal arteries on account of their lack of external support. Mester records a case which followed a kick in the abdomen from a horse.

Intrahepatic aneurysm is exceptionally rare, and cases previously recorded have been associated with suppuration of the liver. Dr. Rolleston points out that the pathology of the formation of aneurysms under this condition is similar to the manner in which aneurysms of the pulmonary artery are found in phthisical cavities in the lungs, viz. as a result of softening of the coats of the vessel from an external inflammation, associated with destruction of the tissue of the organ, leading to a loss of the local external support of the artery.

The main vessel, the right or left branch or smaller branches in the liver may be involved. The sac may increase to the size of a cocoa-nut if it arise from the main vessels. Rupture may occur into the peritoneal cavity, bile duct, stomach, duodenum, colon, or gall-bladder.

The symptoms are: pain which may be continuous or paroxysmal and colicky, jaundice from pressure on, and obstruction of the bile ducts, hæmatemesis, and melæna, if leakage occur into the bile duct. If the sac rupture into the peritoneal cavity collapse and sudden death ensue; if into the stomach or duodenum collapse and severe hæmatemesis soon followed by death. The portal vein may be pressed on and ascites result, but this is uncommon.

A pulsating tumour may be felt in the epigastrium or right hypochondriac region, and on auscultation a systolic murmur may be heard. It would be almost impossible to distinguish an

aneurysm of this artery from one of the cœliac axis or abdominal aorta. When jaundice is the prominent symptom it would be difficult to exclude a diagnosis of gall-stones, especially if it be associated with attacks of pain resembling biliary colic.

ANEURYSM OF THE ARTERIES SUPPLYING THE STOMACH AND OF THE CYSTIC ARTERY

These are also extremely rare, and may be due to embolism and to primary arterial disease. Only one case of this nature was found in the Guy's Hospital records. The patient was a man, æt. forty-seven, who had been suffering for nearly two years from swelling and œdema of the legs. He was found to have a very hypertrophied heart with aortic incompetence. He died suddenly. At the post-mortem examination the abdominal aorta was found to be very atheromatous. The peritoneal cavity contained a large quantity of blood clot and blood-stained serum. An aneurysm the size of a pigeon's egg was found springing from the right gastro-epiploic artery; its surface was eroded and discoloured, and it had ruptured. There were two smaller aneurysms the size of beans, which contained black coagula. A number of small round black bodies were found along the attached margins of the intestine, which might have been mistaken for little varices, had it not been for the aneurysms found elsewhere. There was another smaller aneurysm the size of a pea on one of the omental arteries. The liver was cirrhosed. There were three small nodules containing black coagula, which projected from the surface of the gall-bladder, and two aneurysms of the cystic artery. The heart weighed $23\frac{1}{2}$ ozs. The aortic valves were incompetent. There were no vegetations nor ante-mortem thrombi in either the left ventricle or the left auricle. There was no evidence to show that these aneurysms were the result of embolism. The atheromatous condition of the aorta pointed to a primary arterial degeneration as the cause.

An aneurysm may arise in the floor of a gastric ulcer, or the sac may bulge into the stomach and ulceration of the mucous membrane over it may take place. Rupture may occur into the stomach.

ANEURYSM OF THE PANCREATIC ARTERY

A case has been described by Babington. The patient was a female aged twenty-five years. For several months before her death she

had suffered from a dull, heavy pain in the epigastric region, which had increased in severity. She was suddenly seized with extreme exhaustion, followed by vomiting and purging of blood, and died in about fifteen hours from syncope. Death was due to hæmorrhage into the duodenum consequent on rupture of an aneurysm of the pancreatic artery into that part of the intestine.

ANEURYSM OF THE SPLENIC ARTERY

Of the four cases of aneurysm of the splenic artery one was intrasplenic, and the result of a septic embolus, the primary disease being infective endocarditis. In this case the spleen weighed 16½ ozs., and contained a large decolorised infarct, in the centre of which was the aneurysm measuring three-quarters of an inch in diameter. One was in a patient, æt. forty-six, who died of phthisis. The splenic artery was very atheromatous and calcareous, and the aneurysm was saccular and about the size of a pea, with marked calcareous walls. Another case was in a patient seventy-seven years of age, who died of strangulated femoral hernia and peritonitis. The splenic artery was atheromatous, and in the hilus two small calcareous aneurysmal dilatations of the artery were found. The cause in these two last-mentioned cases was undoubtedly a primary arterial degeneration. The remaining case was an extraordinary one of aneurysmal varix of the splenic and mesenteric veins. The patient was a woman, æt. forty-nine, whose chief symptoms were severe pain and tenderness in the upper part of the abdomen, vomiting, diarrhœa, hæmatemesis, melæna, and albuminuria. She was thin and wasted. A loud blowing murmur, in character like a venous hum, was heard on the left side over the splenic dulness, extending backwards as far as the spine and upwards to the sixth rib, the point of maximum intensity being in the ninth space in the mid-axillary line. A few days before death she developed parotitis and delirium. At the necropsy the main portal vein was found to be patent. The splenic vein at the hilus of the spleen was dilated into a cavity the size of a hen's egg, and filled with laminated clot, but it was found to have a valvular communication with the splenic artery about two inches from its origin. There were several infarcts in the spleen. The right gastro-epiploic, the coronary, and some branches of the inferior mesenteric arteries were filled with ante-mortem clot.

Dr. Osler has reported a case in a man, aged thirty, whose

chief symptoms were severe epigastric pain, vomiting, occasional hæmatemesis, and hæmorrhage from the bowel, which led to a diagnosis of gastric ulcer. There was a deep-seated non-pulsating tumour in the left hypochondriac region, the dulness of which was continuous with that of the spleen. An aneurysm the size of a cocoa-nut, which had opened into the colon, was found.

Dr. Rolleston (*Path. Soc. Tr.* vol. l., 1899) records a case occurring in a woman aged thirty-seven, who died from the effects of granular kidney, in whom an aneurysm of the size of a cherry developed on an apparently normal vessel, there being no sign of atheroma nor of embolism, and it was suggested that the artery was in a condition of hyperplasia, and that the high vascular tension led to a dilatation of the vessel which is but ill-supported.

ANEURYSM OF THE MESENTERIC ARTERIES

Of four cases of aneurysm of the superior mesenteric artery two were caused by embolism from infective endocarditis, one was associated with aneurysm of the abdominal aorta, and the other with atheroma of the aorta and its branches. It may also be the result of injury or non-septic embolism. The sac may arise from the main trunk in any part of its course, or from one of its branches. It may give rise to a pulsating tumour indistinguishable from aneurysm of the abdominal aorta, but is more likely to be movable. It may press on the bile duct and cause jaundice, on the renal arteries and lead to albuminuria or uræmia, if both vessels are obstructed. It may compress the ureter and cause hydronephrosis and attacks of pseudo-renal colic. Cases have been recorded in which no symptoms were manifested until the fatal rupture, although some pain and discomfort, sometimes excessive, are the more usual conditions. Death may result from rupture into the peritoneal cavity or mesentery. The vessel may become obstructed by a thrombus, or its branches blocked by emboli, and infarct of the corresponding part of the intestine may be produced, causing acute abdominal pain and distension, vomiting, and hæmorrhage.

The inferior mesenteric is even more rarely the seat of aneurysm than the superior branch. Dr. Dreschfeld collected 25 cases in which these vessels were involved, 20 being of the superior and 5 of the inferior mesenteric. It is most frequently the result of embolism. The sac may rupture into the peritoneal cavity, mesentery, or intestine. The chief clinical manifestation of aneurysm of this vessel is hæmorrhage from the bowel.

ANEURYSM OF THE RENAL ARTERY

The case of aneurysm of this artery was the result of septic embolism from infective endocarditis. The sac was about the size of a pea, and arose from a branch of the artery in the substance of the kidney midway between the pelvis and the capsule. Both kidneys and the spleen contained infarcts. Mr. H. Morris collected 21 cases, of which 12 were due to traumatism. The main vessel or one of its branches outside or inside the kidney may be affected. It may press on the hilus of the kidney and cause attacks of renal colic. If the sac become filled with thrombus the kidney may necrose and atrophy. It may rupture into the peritoneal cavity and cause sudden death; into the perinephric tissues and give rise to a large tumour in the loin; or into the pelvis of the kidney and cause hæmaturia. Recognisable tumour, pulsation, and bruit may be absent. A case has been recorded which was mistaken for carcinoma of the kidney and was cured by nephrectomy.

Treatment.—It is very difficult to distinguish an aneurysm of one of the branches of the aorta from an aneurysm of the main vessel, and the treatment is practically the same, depending mainly on the position of the resulting tumour. Many cases of this nature are not diagnosed during life, and may be classed as pathological curiosities.

A case of aneurysm of the spermatic artery has been described by Oliver (*Cincin. Lancet*, 1893, N.S. xxx. 197), and of the phrenic artery by Dean (*St. Louis Med. and Surg. Journal*, 1884, xvi. p. 47).

EMBOLISM AND THROMBOSIS OF THE ABDOMINAL BLOOD-VESSELS

EMBOLISM AND THROMBOSIS OF THE ABDOMINAL AORTA

Complete occlusion of the abdominal aorta from embolism or thrombosis is an extremely rare condition. Professor Welch[1] collected 59 published cases, and there were only 4 in the Guy's Hospital records spread over a period of forty-seven years. It is most frequently associated with chronic cardiac and arterial disease, particularly mitral stenosis. Twenty of Welch's collection, and two of the Guy's Hospital cases were due to this cause. It has

[1] Allbutt, *System of Medicine*, vol. vi. 273.

also been noticed in connection with fibroid heart, acute or infective endocarditis, ante-mortem clotting in the left ventricle, atheroma aneurysm and compression of the aorta, and certain acute diseases, such as erysipelas, pneumonia, typhoid fever, and rheumatism.

It is often difficult to say whether embolism or thrombosis is the primary condition. The aorta just above the bifurcation is the part almost invariably plugged, and the common iliac arteries are usually occluded, the thrombus sometimes spreading into the femorals and even to the tibials. In 21 of Welch's cases the thrombus did not reach as high as the inferior mesenteric artery, and only in one did the clot extend to the level of the cœliac axis. Branches of the abdominal aorta may be occluded by independent thrombi or emboli, and the common occurrence of infarcts points to embolism as the most frequent primary cause of the plugging.

The chief effects of occlusion of the lower end of the aorta are: paraplegia and gangrene of the legs; if the inferior or superior mesenteric be plugged, infarction of the intestine; and if the renals be occluded, infarction and necrosis of the kidneys and uræmia.

The **symptoms** are: paraplegia coming on rapidly with pain in the legs, or sudden loss of power, more marked in one leg if the corresponding common iliac artery be occluded more completely than the other, pain in the legs, loins, and lower part of the abdomen, which is often excruciating and paroxysmal in character. These symptoms may, however, come on more gradually with tingling and numbness in the legs followed by pain and paraplegia. Gangrene of one or both of the legs, commencing in the toes and rapidly spreading upwards; albuminuria, diarrhœa, hæmorrhage from the bowels, tympanites, hæmaturia, hæmatemesis, and priapism are symptoms which sometimes occur. The muscles may be rigid or flaccid. There may be numbness, anæsthesia, involving the lower part of the legs, analgesia, or hyperalgesia. The legs become cold and may be œdematous. If the paralysis be complete the reflexes are abolished. Bed-sores may appear, and there may be retention or involuntary passage of urine and fæces. There is absence of pulsation in the femoral arteries and their branches. There may be tenderness over the lower part of the aorta, and an absence of pulsation in the vessel immediately above its bifurcation. An absence of bleeding on incising the legs has been noted. Gangrene usually rapidly supervenes, and death may occur within twenty-four hours. Fourteen of the patients included in Welch's series of cases died within the first four days. Two

explanations have been offered to account for the sudden onset of paraplegia, viz. ischæmia of the spinal cord, or of the peripheral nerves and muscles. This form of paraplegia is distinguished by its association with absence of femoral pulsation, and of the presence of coldness and lividity of the legs followed by gangrene.

Treatment.—The patient should be put on a water bed, and every precaution taken for the prevention of bed-sores. The legs should be wrapped in cotton wool and kept warm. The diet should be light. Potassium iodide is the drug which is usually administered.

EMBOLISM AND THROMBOSIS OF THE MESENTERIC VESSELS

Of 21 cases from the Guy's Hospital records there were 8 of embolism, and 4 of thrombosis of the superior mesenteric artery and its branches, 8 of thrombosis of the mesenteric veins, and 1 of embolism of the inferior mesenteric artery. The primary cause of the embolism in 5 of the cases was infective endocarditis, in 3 chronic cardiac disease, of which one was mitral stenosis with an ante-mortem thrombus in the appendix of the left auricle. The causes of thrombosis of the mesenteric arteries were appendicitis, enteritis, carcinoma of the liver, and an aneurysm of the abdominal aorta containing adherent thrombus. Thrombosis of the mesenteric vessels sometimes follows abdominal operations, and not necessarily involving the intestines (*Maryland Brit. Med. Jour.* 1901, p. 1454).

Infarction of the intestine, a condition which may be followed by necrosis and peritonitis, is the chief effect of the sudden occlusion of the superior mesenteric artery or one of its branches. The portion of intestine involved becomes dilated, its walls swollen, œdematous, and thickened from engorgement and effusion of blood; it assumes a dull, purplish, red appearance with a sharp margin of demarcation marking it off from the adjacent healthy bowel. The peritoneum covering the infarcted intestine becomes dull and lustreless, and is soon covered with flakes of lymph. The mucous membrane is thickened and infiltrated with blood, becomes a deep purple or dark brownish purple colour, and is covered with patches of a dirty brownish gray, adherent, diphtheritic-like membrane. The gut may contain dark or light blood, and the peritoneal cavity may also contain a good deal of blood-stained serous fluid. The bowel may become gangrenous and perforation may occur, leading to general peritonitis. The mesentery and mesenteric glands may become swollen and hæmorrhagic.

According to the experiments of Welch and Mall, the blood which produces infarction comes from the anastomosing arteries, and not by reflux from the veins. If the plugging be gradual no infarction results, as there is time for a collateral circulation to develop. In a case in which gradual blocking of the superior mesenteric artery had occurred as a result of the formation of a large adherent thrombus in the sac of an aneurysm arising from this part of the aorta, the circulation had been carried on by a large tortuous vessel running down in the position of the colica transversa, and anastomosing with the colica sinistra of the inferior mesenteric artery, which vessel was abnormally large.

The **symptoms** of sudden complete occlusion either from embolism or thrombosis are:

An extremely sudden attack of severe, paroxysmal, colicky pain associated with tenderness in the region of the umbilicus, symptoms which are probably due to the violent and sudden spasm of the bowel which immediately follows the plugging of the vessel. The spasm is followed in a few hours by paralysis and consequent distension of the intestine, the meteorism sometimes being extreme. Vomiting is another early and important symptom; the vomit may at first consist of stomach contents, it then may become bile-stained, bloody, and even stercoraceous in character. Blood is usually passed from the bowel, and it may be dark, or the motion may be loose and contain bright red blood and flakes of mucus. The abdomen becomes distended and tympanitic, the pulse rapid and feeble, the temperature subnormal, the extremities cold and clammy; finally collapse supervenes, and the patient dies from cardiac failure. A tumour has occasionally been felt when the mesentery is much thickened from infiltration with blood. Death usually occurs in two or three days.

Thrombosis of the *veins* was found associated with empyema, appendicitis, hernia, cirrhosis of the liver, carcinoma of the mesenteric glands, angina Ludovici, and aneurysmal varix of the splenic vessels. The superior mesenteric vein and its branches are much more frequently affected than the inferior mesenteric. The thrombus may spread upwards from inflammatory lesions of the intestine, or downwards from thrombosis of the portal or splenic veins. If one of these veins be suddenly occluded the symptoms are much the same as with plugging of the mesenteric artery, but may be more acute and severe. If the thrombus be of slow formation there may be no symptoms. The changes in the intestines are also similar to those produced by embolism or thrombosis of the arteries.

Diagnosis. — The extremely sudden onset with vomiting, abdominal pain, and tenderness is most likely to lead to a diagnosis of acute intestinal obstruction, acute peritonitis, or acute hæmorrhagic pancreatitis. Hæmorrhage from the bowel is extremely rare in any form of acute intestinal obstruction, except acute intussusception, only two or three cases of hæmorrhage as a result of volvulus of the small intestine having been recorded. The presence of a cylindrical shaped abdominal tumour would be much in favour of intussusception, but it must be borne in mind that a tumour may be felt in cases of occlusion of the mesenteric vessels if the mesentery be thickened from effused blood.

If the patient be found to be suffering from any form of chronic cardiac or arterial disease, or any other condition which is likely to give rise to embolism, and if any signs of embolism in any other parts of the body be manifest, the diagnosis of occlusion of the mesenteric vessels should not present very great difficulty.

The **prognosis** is very grave if the occlusion be complete and lead to gangrene.

Treatment.—Medical treatment is of no avail. Two cases have been recorded in which the infarcted intestine has been successfully resected; and as it has hitherto been such a fatal disease, laparotomy should be performed, and the intestine examined to see if resection be possible.

EMBOLISM AND THROMBOSIS OF THE RENAL VESSELS

Embolism of branches of the renal arteries is of common occurrence in endocarditis, and leads to the formation of infarcts. Emboli may be derived from vegetations on the mitral and aortic valves, ante-mortem thrombi in the appendix of the left auricle and apex of the left ventricle, pieces of atheromatous pulp and calcareous material from atheromatous ulcers of the aorta, and ante-mortem thrombi in aneurysmal sacs.

It is rare to find one of the main renal arteries blocked, but when it does occur almost complete necrosis of the kidney follows, only a thin outer zone of healthy cortical substance remaining, on account of the collateral supply of blood from the capsular arteries. In a case of aneurysm of the abdominal aorta in which the left renal artery had been completely occluded a small relic only of the left kidney was found, which appeared to consist of a shrivelled pelvis, some vessels, and small cysts, no distinct kidney substance being visible.

The infarcts which follow embolism of smaller branches of the renal arteries are usually pale and wedge-shaped, and the subsequent changes which occur are dependent on the nature of the emboli. If they contain pyogenic micro-organisms multiple abscesses may follow, but a more frequent result is cicatrisation, which leads to the formation of depressed cicatrices and a certain degree of atrophy of the kidney. In infective endocarditis the kidney presents a very typical appearance, the cortex being pale and speckled with numerous small hæmorrhages due to embolic plugging of minute arteries by aggregations of micro-organisms (see p. 576).

Small emboli do not exhibit any obvious or characteristic **symptoms**. Large infarcts project from the surface of the organ, stretch the capsule, and in consequence cause a good deal of pain and tenderness in the back and loins, and also give rise to hæmaturia and albuminuria. The amount of blood which is voided in the urine is usually small, and is characterised by its sudden onset and equally sudden disappearance.

Embolism of the renal arteries has to be differentiated from other conditions which cause symptoms similar to those mentioned above. On account of its frequent association with heart disease it may be difficult at times to say whether the symptoms are due to congestion or embolism, but if associated with painful enlargement of the spleen and signs of embolism elsewhere it would point to the latter condition. Absence of anasarca is an important but not conclusive indication that the symptoms are not due to acute nephritis. The pain is not so severe, nor is it colicky as in renal calculus.

Thrombosis of the renal artery or its branches will produce changes similar to those which result from embolism. It is, however, a much rarer occurrence. Of 7 consecutive cases from the Guy's Hospital records, 2 were associated with cardiac disease, mitral stenosis, and fibroid heart, no ante-mortem thrombi being found in the left auricle and ventricle; 3 with aneurysm of the abdominal aorta; 1 with lardaceous disease; and 1 associated with generalised thrombosis of the aorta, middle cerebral and splenic arteries, and the portal and hepatic veins. It may be associated with endarteritis.

Thrombosis of the renal veins is much commoner. An analysis of 26 consecutive cases from the Guy's Hospital records showed that 14, or more than 50 per cent, were associated with malignant disease, of which 8 were of the kidney, the remaining 6 being of

the sigmoid flexure, mesenteric and lumbar glands, and the bladder; 3 with adjacent suppuration; 2 with abdominal aneurysm; and 1 each with caseous mesenteric glands, tuberculous kidney, injury, acute nephritis, lardaceous kidney, broncho-pneumonia, and strangulated hernia. It may occur primarily in certain acute diseases, *e.g.* influenza, diphtheria, epidemic diarrhœa, etc. The thrombosis may extend from the inferior vena cava, especially when the cause is some septic condition of the legs or pelvic organs; in the case of growth in the kidney the thrombosis may commence in the renal veins and extend into the inferior vena cava. When due to malignant disease the thrombosis itself may become malignant. If both renal veins become occluded suppression of urine may follow.

The **prognosis** is bad when the primary cause of the trouble is infective endocarditis, malignant disease, or sepsis.

There is no special **treatment** which can be directed to the local condition except for the relief of pain.

THROMBOSIS OF THE INFERIOR VENA CAVA

Of 72 cases collected from the Guy's Hospital records 36, or 50 per cent, were associated with malignant disease, 11 with suppuration in the liver, femur, hip joint, psoas and iliacus muscles, etc., 8 with tuberculous lesions of the spine and mesenteric glands, 4 with the puerperal state, 4 with chronic cardiac disease, 2 with septic uterine diseases, 2 with erysipelas, 1 with typhoid fever, and 1 with spleno-medullary leukæmia. Of the 36 which were due to malignant disease the primary growth was in the stomach in 10, and in the kidney in 9. The thrombus may be caused by the growth itself, or by glands enlarged from secondary deposits pressing on and in many cases invading the main vessel. When the primary growth is in the kidney the renal vein is first blocked by a thrombus which gradually spreads into the inferior vena cava until the latter may become completely occluded. In a case of this nature the whole of the inferior vena cava was blocked by a thrombus, which had itself become infiltrated with malignant disease, and had extended upwards and bulged into the right auricle. The condition has also been known to follow accidents, particularly falls from a height. The greater number die suddenly from pulmonary embolism, the upper part of the clot becoming detached.

The chief symptoms are swelling and œdema of the legs and lower part of the back and abdomen, dilatation and tortuosity of the veins over the upper part of the thighs, groins, loins, abdomen, and lower part of the thorax. In some cases the veins are enormously dilated, and may measure a centimetre or more in diameter. The blood flows in an upward direction in order to reach the right auricle through the superior vena cava. The direction of the flow of blood may be determined by emptying a portion of one of the veins by pressing it with the finger and then by drawing the finger downwards over it, the pressure being still retained, when it will be seen that the part of the vein which has been pressed on will remain empty for a time. If, on the other hand, the finger were drawn in an upward direction the vein would immediately fill below the finger as soon as the pressure was relaxed. If the renal veins be obstructed there may be albuminuria and hæmaturia, but these symptoms are by no means constant. In the case above mentioned, where the main trunk first became plugged at the level of the renal veins, œdema of the legs developed, and this was followed a short time afterwards by ascites, when the thrombus had increased in size upwards, and had occluded the hepatic veins. The collateral circulation which is usually developed is by means of the superficial epigastric, the superficial circumflex, the external pudic, the long thoracic, the lumbo-vertebral anastomotic vein of Braune and other small superficial veins.

Occasionally complete plugging may occur without any marked symptoms, when, for some reason, probably because the iliac veins are not occluded, the collateral circulation is carried on by the intra-abdominal veins alone. In a case of this kind, in which the lumen of the inferior vena cava was completely obliterated, there was a bunch of enormously dilated and varicose veins in front of the left ovary, passing into the left iliac fossa and ending in the left ovarian vein, which was about the diameter of an ordinary lead pencil, to finally terminate in the left renal vein. The iliac veins were pervious, and it appeared that the blood from the lower limbs had passed into the internal iliac veins, and so to the side of the uterus, along the lateral margin of which a large vein ran, united by a kind of plexus behind the cervix. There was also a large vein on the right side connected with the ilio-lumbar vein, which ran into the perinephric tissues, and entered the upper border of the right renal vein. The epigastric veins were not in the slightest degree dilated. The chief diagnostic sign of this

condition is the enormous dilatation of the veins over the abdomen with the blood taking an upward direction.

When resulting from accident the first symptoms have been sharp pain and paræsthesiæ in the lower limbs, with marked diminution of sensibility and power of movement.

Treatment.—The patient should be kept as quiet as possible in bed for at least five or six weeks, and should avoid any sudden movement or jerk. The abdomen should not be palpated. The diet should be light. Iodide of potassium may be administered internally.

Thrombosis of the Portal Vein

adhesive or non-suppurative pylephlebitis

This condition is brought about by those pathological changes which cause a slowing of the blood stream in the portal vein or its branches. Of 32 cases from the Guy's Hospital records, 8 were associated with cirrhosis of the liver, 7 with carcinoma of the stomach, 5 with carcinoma of the rectum or sigmoid, 2 each with injury to the abdomen, chronic cardiac disease, primary carcinoma of the liver, and carcinoma of the pancreas, and 1 each with carcinoma of the ovary, carcinoma of the kidney, splenic anæmia, gall-stones, intestinal obstruction, and ulceration of the colon. In addition to these causes it has been found to be produced by syphilis, perihepatitis, extension of the thrombus from the tributaries of the vein, infectious diseases, marasmus, and primary calcification of the walls of the vessel. The vein is frequently found to be compressed by tumours or enlarged portal glands. The thrombus may completely block the main vessel, and become adherent and organised. Its frequent association with cirrhosis of the liver has led to a difference of opinion: whether the cirrhosis be the result or the cause of the thrombosis. The chief pathological changes produced by it are: dilatation of the superficial veins of the abdominal wall, varicose œsophageal veins, congestion and hæmorrhagic erosion of the mucous membrane of the stomach, congestion of the intestines, hæmorrhoids, and an enlargement of the spleen which may be considerable.

The collateral circulation opened up, as a result of complete occlusion of the portal vein, is the same as that in cirrhosis of the liver.

The **symptoms** and **physical signs** are: vomiting, hæmate-

mesis, diarrhœa, melæna, enterorrhagia, progressive weakness, ascites, enlargement of the spleen, and dilated superficial veins with the blood coursing upwards. A murmur which may be systolic in rhythm or a continuous venous hum may be audible over the spleen.

The ascites may have an extremely rapid onset, and another characteristic feature of this disease is the rapidity with which the ascites returns after the fluid has been removed by paracentesis. It has to be distinguished from the other conditions which may cause portal obstruction, but the most important diagnostic sign is the acute onset of ascites and its rapid recurrence after operation.

The **treatment** is the same as that for cirrhosis of the liver.

SUPPURATIVE PYLEPHLEBITIS

This is a very obscure disease, and one which is not as a rule diagnosed during life. It is of great rarity, the post-mortem records of Guy's Hospital showing 20 cases only out of 17,474 necropsies performed during a period of forty-three years. It is an inflammation of the portal vein resulting in the formation of suppurating thrombi and pus, with multiple abscesses in the liver. It is due to a septic lesion of one of the organs drained by the tributaries of the portal vein, or more exceptionally to a lesion of the portal vein itself, or of the liver involving one or more of its branches. There may be a history of or evidence of the original local lesion followed by the development of symptoms of pyæmia, which may, possibly, be associated with signs of disease of the liver.

Etiology.—Appendicitis is by far the most frequent cause, nearly half the cases resulting from it. The appendicitis is often latent, the history of the attack, or of previous attacks, indefinite, and the typical physical signs absent, so that it is frequently overlooked, and even at a necropsy, unless carefully sought for, the primary lesion in the appendix vermiformis may be missed. Other causes are: gastric ulcer, duodenal ulcer, dysenteric ulceration of the colon, sloughing of the cæcum, ulceration of the rectum, thrombosis and suppuration following operations for hæmorrhoids, submucous abscesses of the rectum implicating the hæmorrhoidal veins, inflammation of the umbilical vein in infants, a fish bone perforating the superior mesenteric vein (Frerichs), gall-stones, and suppurating gall-bladder, ulceration of the common bile duct extending to the portal vein, hepatic abscess or suppurating hydatid

cysts involving the hepatic branches of the portal vein, abscess of the spleen, suppurative pancreatitis, suppurating mesenteric glands, pyosalpinx, suppurating ovary, abortion, and localised peritonitis following injury. One of the most extraordinary facts in connection with the etiology of this disease is the absence or extreme rarity of typhoid ulceration as a cause, seeing that it is by far the commonest form of ulceration of the intestine met with in this country. There does not appear to be any particular reason for this exception, for the typhoid bacillus may be found not only in the ulcers, mesenteric glands, and spleen of patients suffering from typhoid fever, but also in abscesses resulting from periostitis, perichondritis, rupture of muscles, and in suppuration of the gall-bladder. When it does occur as a complication of typhoid fever it is almost invariably found to be secondary to suppuration in connection with ulceration of the appendix vermiformis. The comparative rarity of the affection in the disease known as ulcerative colitis is also noticeable.

Morbid anatomy and pathology.—The body is usually much emaciated. The portal vein, its tributaries, and branches may be inflamed, and as a result dilated and filled with thick greenish or yellowish foul-smelling pus, or a mixture of pus and blood, or of soft suppurating partly adherent thrombi, bathed in or channelled by pus. The walls of the veins are often thickened and softened, and, in the liver, partly detached from the surrounding tissue. The intima may be irregular and shaggy from adherent lymph. In some cases a track of suppuration can be traced within the veins from the seat of the primary lesion to the portal vein, and even to its smallest branches within the liver; in others the tributaries of the portal vein contain neither pus nor thrombi; and in others the change is limited to one of its main branches, or even to the smaller branches in the substance of the liver. In more than 50 per cent of the cases the liver is slightly and uniformly enlarged, and is frequently firmly attached by means of fibrous adhesions to the under surface of the diaphragm as a result of the inflammatory process spreading to the surface and causing perihepatitis. The surface of the organ is generally smooth, but the small abscesses may project and form groups of small yellow nodules, covered with flakes of recently deposited lymph. On section abscesses varying in size and distribution are invariably found. The whole or only part of the liver may be studded with small areas of suppuration smaller than peas, and larger abscesses may result from several of these smaller foci of suppuration running together. The right lobe

may be more affected than the left, and *vice versa*, or the abscesses may be confined to a limited area supplied by a comparatively small branch of the portal vein. On squeezing a section of the liver pus may be seen to well up and exude from the portal canals. The dilated portal vein itself or abscesses connected with its main branches may open into the cystic or hepatic ducts, and in consequence the gall-bladder may become distended with pus. Another complication is the formation of local abscess in the portal fissure, or in the neighbourhood of the pancreas. Suppurative peritonitis occurs in about half the cases. The spleen is not invariably enlarged, but is soft and pulpy, and may contain abscesses. The capsule is generally thickened as a result of old or recent capsulitis. The pancreas is healthy unless it happens to be the seat of the primary septic focus.

Pleurisy involving the upper surface of the right half of the diaphragm and the base of the right lung is a very common complication, and it may be associated with the presence of clear serous fluid or pus in the pleural cavity.

Histological examination of the liver shows multiple abscesses of varying size, and the portal capillaries and branches of the portal vein distended with pus. Colonies of bacilli or of cocci are found in the pus within the vessels, and also in the abscesses in the hepatic tissue, in the neighbourhood of which the hepatic cells are undergoing necrosis, and there is small-celled infiltration. The micro-organism most frequently found is the bacillus coli communis, but the streptococcus pyogenes, the staphylococcus pyogenes aureus, or the bacillus pyocyaneus may also be present. The disease is practically a local pyæmia, and is due to an invasion of the portal vein, its tributaries or branches, by pyogenetic micro-organisms, which gain access through a septic lesion of one of the organs drained by the portal system, most frequently the appendix vermiformis, or by direct implication of the portal vein itself, or one of its branches in the substance of the liver. As a result of such lesions the small vein draining the area affected is invaded by the micro-organisms; phlebitis, thrombosis, and suppuration of the thrombus follow, and from this focus the portal vein and its branches may be infected by direct extension or by embolism. In the former cases there would be a continuous track of pus and suppurating thrombus from the site of the primary septic change to the multiple abscesses in the liver, and in the latter case the veins leading from the primary focus, and the portal vein itself, might be free of pus and thrombus, and the abscesses might be limited to

certain portions of the liver corresponding to the branches of the portal vein infected by the emboli. When a branch of the portal vein in the liver is infected by some adjacent septic lesion the phlebitis, thrombosis, and suppuration may extend towards the portal capillaries on one side and towards the main portal vessels on the other. Whatever may be the cause of the condition when the smaller branches of the portal vein are inflamed and contain pus and suppurating blood clot, the inflammatory process spreads to the adjacent hepatic tissue and leads to small-celled infiltration, necrosis of the liver cells, and consequent abscess formation. If the spleen be enlarged it is due to a toxic condition of the blood rather than the passive congestion, as the portal vein is never completely obstructed by a thrombus in this disease.

Symptoms and physical signs.—The onset may be acute, the initial symptoms being vomiting, rigors, pain in the right side of the abdomen, chest, back, or loins, and diarrhœa; on the other hand it may be indefinite and insidious, as in typhoid fever, beginning with general malaise and headache. Pyrexia, pain in the chest or abdomen, jaundice, vomiting, and diarrhœa may be prominent early symptoms. After pyrexia, pain is one of the most important and constant symptoms; it is usually situated in the right hypochondriac region, but may be in the epigastric, umbilical, or left hypochondriac regions; vomiting and diarrhœa are usually present, but the latter condition may be replaced by constipation. Sweating and rigors are not so common as many text-books would lead one to suppose, for the latter symptom only occurs in about half the cases. Jaundice is another symptom which is not invariably present, for it only occurs in about 55 per cent of the cases. In the later stages of the disease delirium, collapse, and coma may supervene. A rise in temperature is by far the most constant symptom, but it does not run any particular course, for it may be continuous in character and resemble pneumonia, or it may be typhoidal in character, or remittent or intermittent. Hyperpyrexia may occur. The pulse is rapid, soft, compressible, and running, and above the physiological ratio to the temperature. The respiration is increased, which is due chiefly to the frequency with which the right pleura may become affected. When the disease is well advanced the patient lies on his back, appears weak, prostrated, wasted, and anæmic, and looks very ill. The eyes are sunken, and the expression is pinched and anxious. The cheeks may be flushed and the complexion may have a muddy appearance. The skin may be hot and dry, but is usually

moist. The lips and teeth are covered with sordes, and the tongue is dry and furred.

The abdomen may or may not be distended. A slight fulness or even a marked swelling may be noticed in the right hypochondriac and epigastric regions. An increased sense of resistance or marked rigidity may be appreciated in these regions on palpation. The liver can usually be felt to be enlarged, and its edge may be detected just below the costal margin in the right nipple line, and occasionally as low as the umbilicus. The edge is sharp and well defined, and the surface is nearly always smooth and regular, but small, soft, fluctuating nodules may rarely be felt when the abscesses are sufficiently large to project from the surface of the organ. There is, as a rule, pain and tenderness on palpating the epigastric or right hypochondriac region, whether the liver be enlarged or not. The spleen may be felt below the costal margin; if a splenic abscess be the primary cause of the disease, it is tender, a fluctuating swelling may be felt, and a friction rub heard over it on auscultation. Pain, tenderness, and a tumour, or increased sense of resistance may be felt in the right iliac fossa if the condition be secondary to appendicitis. The lower part of the thorax on the right side may show distinct bulging, and the intercostal spaces may be filled out—conditions due to perihepatitis, and consequent adhesions fixing the liver to the diaphragm and thoracic wall. A pleuritic rub may be heard over the lower part of the right side of the thorax, or the signs of fluid may be present. The urine may be normal, or it may contain albumin or bile pigment, and the chlorides may be diminished. The stools may be steel-gray coloured, typhoidal in character, light clay coloured, or dirty white. The blood shows a diminution of hæmoglobin and red blood corpuscles, and a polymorphonuclear leucocytosis. The duration of this disease is extremely variable, the average being about 6 weeks. Goodhart has recorded a case which lasted for 296 days, and at the necropsy signs of healing and cicatrisation of the abscesses in the liver were found. The most rapidly fatal case reported lived for 5 days and 3 hours from the onset of the first symptom.

The **prognosis** is extremely grave. There is no authentic case of recovery from this disease on record.

The **diagnosis** presents great difficulties, and in the majority of cases is not made during life. It has been mistaken for typhoid fever, pyæmia, septicæmia, pneumonia, empyema, hepatic abscess, subdiaphragmatic abscess, catarrhal and other forms of jaundice, cirrhosis of the liver, gastro-enteritis, spinal caries, and infective

endocarditis. Pyrexia, rigors, sweating, emaciation, prostration, sallow earthy complexion, jaundice, abdominal pain, tenderness in the right hypochondriac or epigastric regions, together with slight uniform enlargement of that organ, especially if associated with or following any ulcerative lesion of the alimentary tract below the œsophagus, should suggest the possibility of suppurative pylephlebitis. The chief points, therefore, on which a diagnosis should be base dare: the recognition of a condition of pyæmia; the absence of a cause on the venous side of the circulation or of infective endocarditis; the presence of a focus of infection within the abdomen, together with signs of implication of the liver.

Typhoid fever may be distinguished by the pulse, temperature, and respiration ratio, for in the majority of cases of this disease the pulse ratio is often extraordinarily low, whereas in suppurative pylephlebitis it is higher than the physiological ratio, *e.g.* with a temperature of 104° the physiological ratio of the pulse is 125; in a case of typhoid fever it was 80, and in a case of suppurative pylephlebitis 140. The spleen may be enlarged and the abdomen may be distended in both diseases. Jaundice, sweating, and rigors are exceedingly rare in typhoid fever, but the presence of a characteristic rash would point to this disease. The examination of the blood is of the greatest importance, for a positive Widal reaction would point to typhoid fever or a previous attack of the disease, and, further, the blood in typhoid fever is characterised by leucopenia, whereas in suppurative pylephlebitis there is marked polymorphonuclear leucocytosis.

The frequency with which the right pleura is affected may lead to a diagnosis of primary disease of the pleura or right lung, but a careful attention to the above-mentioned points, and the knowledge of the possibility of such an obscure disease as suppurative pylephlebitis should lessen the chance of it being overlooked.

The **treatment** is unsatisfactory, but must be directed to the relief of symptoms. The only possible hope of a cure would be an early diagnosis, not only of suppurative pylephlebitis, but also of its cause, followed by an immediate eradication of the primary lesion, especially if it should happen to be appendicitis. The possibility of such a serious complication, although remote, should be considered in all cases of appendicitis, and is an additional argument for the removal of the diseased appendix vermiformis after the subsidence of an acute attack.

J. H. Bryant.

DISEASES OF THE KIDNEYS

General Anatomy of the Kidneys

The kidneys lie behind the peritoneum, opposite the twelfth dorsal, the first, the second, and the upper half of the third lumbar vertebræ; the right kidney is a little lower than the left. Each one usually measures 4 inches in length, $2\frac{1}{2}$ in breadth, and $1\frac{1}{2}$ in thickness, and the average weight of the organ is $4\frac{1}{2}$ ounces, the left being a little the heavier. They rest posteriorly on the twelfth rib (occasionally on the eleventh), the diaphragm, and the anterior layer of the lumbar fascia. The pleural cavity descends behind the kidney—the diaphragm intervening—to the extent of approximately the junction of the upper and middle thirds of the kidney.

The anterior surface of the right kidney has the liver, the second portion of the duodenum, the ascending colon and hepatic flexure, and some convolutions of the small intestine in relation with it. The duodenum and the colon are in direct contact with the viscus, without the intervention of peritoneum, but the hepatic and intestinal areas of the kidney are covered by the serous membrane. The stomach, spleen, pancreas, small intestine, and descending colon are in contact with the anterior surface of the left kidney, the pancreas and colon without the intervention of peritoneum. The hilum of the kidney occupies the middle third of the inner border, and is opposite the first lumbar spine. The outer border of the kidney lies 4 inches from the middle line, and the inner border $1\frac{1}{2}$ inches. The upper border of the kidneys is marked approximately by a horizontal line drawn at the level of the eleventh dorsal spine and the lower border by a similar line at the level of the third lumbar spine, the right being rather lower than the left (see table, p. 32).

The renal artery divides on entering the hilum into four or five branches, and lies between the renal vein anteriorly and the ureter posteriorly.

The kidney, enclosed in a fibrous and elastic capsule which contains some plain muscular fibres and in normal organs strips off readily, is embedded in a mass of adipose tissue that assists in maintaining it in position. This fat is traversed by bundles of firm fibrous tissue, and these, with the peritoneum and the tonic contraction of the abdominal muscles, are the main factors, besides the pressure exerted by the neighbouring viscera, in keeping the kidneys in their normal position.

The cortex of the kidney is about 4 mm. in width, but it also extends between the pyramids (usually twelve in number) as far as the sinus of the organ. The branches of the renal artery form incomplete arches between the cortex and the medulla, and these give off other branches known as interlobular arteries that pass up towards the periphery and supply the glomeruli. The pyramids are supplied in part by vessels (the arteriæ rectæ) given off from the arterial arches, and in part by the efferent vessels of the more centrally situated glomeruli forming the so-called false arteriæ rectæ. The convoluted tubules of the cortex are supplied by the efferent vessels of the glomeruli.

The tubules commence in the spherical dilatation that encloses the capillary loop forming the Malpighian tuft, and they consist of a basement membrane lined by an epithelium that varies in its structure in different parts. The Malpighian capsule itself is lined by a layer of flattened cells that are also reflected over the capillary tuft; this epithelium becomes cubical at the commencement of the convoluted tubule. The convoluted tubule is lined with fibrillated or "rodded" cells, the protoplasm of these consisting largely of rods or fibrils placed vertically to the basement membrane. The descending limb of Henle's loop is lined by low flattened cells with clear protoplasmic contents. In the ascending limb the cells are smaller, but otherwise possess similar characters to the rod-like cells of the first convoluted tubule, and this characteristic is still more marked in the continuation of the ascending loop, which is known as the irregular tubule. The epithelium of the second convoluted tubule presents a peculiar highly refractile appearance, and that lining the collecting tubule consists of cubical cells with clear protoplasm.

There is a small amount of interstitial connective tissue between the renal tubules which is most abundant near the papillæ, and is rather more fibrous in character round the Malpighian capsules and the larger blood-vessels

The Physiology of the Kidneys

The kidneys are the glands by which the largest amount of water and of the products of nitrogenous metabolism are excreted. Some of the constituents of the urine besides the water are also eliminated by the skin, the intestines, and the lungs, but approximately half the fluid ingested is excreted under normal conditions by the kidneys.

The substances found in the urine in health and disease are for the most part bodies produced by the metabolism of the tissues, or else substances elaborated by the activity of micro-organisms, or simply introduced from without. Most of the normal urinary constituents are materials which are no longer useful, and some of them possess definite toxic actions especially in large doses. The urinary pigments afford an

instance of the excretion of substances which have no longer any part to play in the economy, and the nitrogenous extractives and many of the salts are substances which, at any rate, in large quantities are distinctly toxic in their action.

Most of the constituents of the urine are instances of a true excretory activity on the part of the organ, that is to say, they are present in the blood going to the kidney, and are simply removed by the activity of the glomerular or the tubular epithelium. In this respect the function of the kidney is in marked contrast to that of many secreting glands where the gland protoplasm actually manufactures the constituent or constituents of the particular secretion concerned. In the case of the kidney this is not so, at any rate as regards most of the important constituents. The kidney does not make urea, but simply removes the urea brought to it in the blood of the renal artery. As regards some components of the urine, and especially uric acid, it has been asserted that the part played by the organ is a more important one, and that uric acid is actually elaborated by the activity of the renal protoplasm (Vol. II. p. 206). This, however, is very doubtful, and it is at any rate probable that the mechanism for the formation and excretion of uric acid is much the same as that in the case of urea. There are, however, some substances which are unquestionably formed in the kidney, and one of the most striking illustrations of these is hippuric acid. This may be present in traces in the normal urine, but is greatly increased in amount after the ingestion of benzoic acid, and numerous observers have shown that the synthesis of hippuric acid by the union of benzoic acid with glycin can be effected by the kidney. There is, therefore, definite experimental evidence that some synthetic metabolic processes can occur in the organ.

The discovery that many glands are provided with an internal secretion led to the conception that the kidney, in addition to its external secretion, has also an internal one; this view was especially advocated by Brown Séquard. The belief in the existence of an internal renal secretion was largely based on the statement that the results seen after the removal of both kidneys were profoundly different to the effects produced by the ligature of both ureters, and it was supposed that in the one case the toxic effects produced were simply dependent on the cessation of the external secretion, and in the other on the cessation of both the external and the internal. This view seemed to derive some support from the results of human pathology, in that it has been long known that the symptoms seen in complete suppression of urine dependent on calculous obstruction are entirely different in their nature and characters from those seen in the suppression accompanying acute or chronic destructive diseases of the kidney. Lastly, some observers, and especially Brown Séquard, claim to have obtained promising results from the treatment of uræmia with renal extracts. Internal secretions

are well marked in the case of several glands, such as the liver and the pancreas, which are provided with ducts and have important external secretions, so that on *a priori* grounds there is no reason why the kidney, notwithstanding its paramount excretory activity, should not have an internal secretion. The evidence, however, in favour of this, as will be seen below, is extremely doubtful.

The functions of the kidney may, therefore, be classified as (1) the excretion of water; (2) the excretion of urea, pigments, extractives, salts; (3) its synthetic activity; and (4) its influence on metabolism.

(1) *The excretion of water.*—It is remarkable that notwithstanding the profound differences in the composition of the urine in mammals and birds, the one being very dilute, the other almost semi-solid, that the minute structure of the kidney of these two classes of animals is not very different, and that the glomeruli which are mainly concerned in the excretion of water are well marked, not only in mammals, but also in birds. Experiments on the amphibia, especially the frog, where the glomeruli have a blood-supply distinct from that of the tubules, have shown that the water is excreted mainly by the glomeruli. The quantity of water excreted necessarily depends in part on the amount ingested and on the amount lost by other channels, especially the skin, the lungs, and the bowel, but its immediate determining cause is the rate of blood flow through the glomerular vessels. The optimum condition is one in which the renal vessels are dilated and those of the body at large constricted, and such an effect may be brought about by the stimulation of the central ends of the posterior roots of the lower dorsal nerves, inasmuch as this causes great dilatation of the renal vessels and a notable rise in blood pressure dependent mainly on the constriction of the vessels in the abdominal area. There is no evidence that such an effect as this may be produced by the action of any drug, although caffein in certain doses causes marked dilatation of the renal vessels accompanied by a slight rise in blood pressure dependent on its action on the heart. Some diuretics produce their effects by causing a local renal dilatation unaccompanied by any general vascular effect, so that the general blood pressure remains at its previous height notwithstanding the renal dilatation. Many of the constituents of the urine produce such an effect as this, and urea has a marked action in dilating the renal vessels. Other substances, such as digitalis, may produce an increase in the amount of urine excreted, notwithstanding the fact that the renal vessels become constricted; the general rise of blood pressure produced at the same time is such, however, that a notable increase in the rate of flow through the renal vessels may occur notwithstanding the renal constriction. The excretion of water is thus intimately associated with the state of the renal vessels, and the kidney is not only extremely vascular, but its blood-vessels are very abundantly supplied with vaso-motor nerves emerging from the lower

dorsal and upper lumbar regions of the spinal cord. There is no experimental evidence pointing clearly to the existence of nerves affecting the excretion of water directly. In other words there are no nerves, the stimulation or section of which is found to produce effects other than those produced through the vaso-motor system. Experimentally, however, many procedures interfere with or diminish the excretion of water without producing, as far as is known, any marked effect on the circulation. Thus the placing of a cannula in the ureter may arrest the secretion of urine, although oncometric observations do not afford any evidence that such an operation produces any effect on the circulation. The passage of a catheter in the human subject will sometimes be followed by complete suppression of urine, which may also be seen as a result of injury or disease in various parts of the body, not directly implicating the kidneys ; under such conditions it is possible that the suppression may be produced through the agency of the nervous system.

The quantity of kidney would also seem to affect the amount of urine secreted. The removal of a portion of one kidney and the whole of the second kidney may be followed by a permanent increase in the amount of urinary water excreted, and this amounts in some cases to twice the normal quantity or even more. No such increase is seen after mere incision and suturing of the organ. It is therefore an effect that is not dependent on any lesion of the nervous system, but is associated with the actual removal of kidney substance. The removal of portions of both kidneys produces the same effect of a permanent increase in the urinary flow, which may amount to as much as four times the normal. It is not clear what the actual mechanism of the increased flow is in these conditions, but it closely resembles the increased excretion of water seen in many morbid conditions in which the quantity of kidney substance is greatly diminished, *e.g.* cystic kidneys, granular kidneys, etc.

(2) *The excretion of urea, extractives, salts, etc.*—Urea and many of the constituents of the urine are excreted through the activity of the epithelium of the tubules ; others, such as sugar in minute quantity, and some of the salts, *e.g.* the chlorides, are excreted by the glomeruli. As already mentioned, these substances are simply removed by the kidney from the blood owing to what is spoken of as a selective activity of the renal epithelium. The percentage of urea in the blood is approximately .015 ; the amount present in the urine is usually 2 per cent, and sometimes considerably more. Sugar is only present in minute traces in the normal urine, notwithstanding the fact that the blood normally contains a far higher percentage of sugar than it does of urea, the average percentage of sugar present being .09.

The constituents of the urine may derive their origin entirely from the food, or else from the metabolism of the tissues, or even in some

cases they may be formed in the alimentary canal, and be subsequently reabsorbed to be excreted in the urine. Such a body as urea is derived from the metabolism of tissue ; such a substance as calcium oxalate is derived entirely from the food, inasmuch as there is no evidence of the formation of oxalic acid during the metabolism of the tissues. Uric acid is in part derived from the food and in part from the body. Some of the urinary pigments and the aromatic sulphates are substances formed in the alimentary canal, and subsequently absorbed and excreted in the urine. It is seen from these facts that the processes that finally determine the composition of the urine are not only very complex, but also of very different origin.

In diseased conditions, where the excretory activity of the kidney is very seriously interfered with, the excretion of urinary constituents may take place by abnormal channels ; thus urea may be excreted in considerable quantity into the stomach, and cases have been described of its excretion in large quantity in the sweat. It is also possible that where the excretory activity of the kidney is seriously compromised toxic materials may accumulate in the intestines and be absorbed in abnormal amount, so causing symptoms of poisoning. The renal epithelium, glomerular and tubular, not only excretes the normal constituents of the urine, but in disease a considerable number of abnormal substances may be present, derived either from an abnormal metabolism or from the presence in the body of toxic substances either ingested or elaborated by the activity of micro-organisms. In a number of diseases micro-organisms may be present in the urine, although it is probable that in these circumstances the physiological integrity of the kidney has been damaged. Enteric fever and acute tuberculosis afford instances where a specific microbe may be found in the urine.

The substances normally excreted in the urine produce, when introduced into the circulation, a considerable dilatation of the renal blood-vessels ; this is especially seen in the case of urea. Thus many of the substances normally excreted may be classified as diuretics, urea, sodium chloride, sugar, etc. The accompanying vascular dilatation leads to the increased secretion of water, and this washes down the tubules the materials that have been excreted by the activity of the tubular epithelium. Although the two processes are normally associated they can occur independently of one another, as in the well-known experiments of Heidenhain, where, after section of the spinal cord in the rabbit, the epithelium of the tubules still excretes sulphindigotate of soda, notwithstanding the greatly diminished excretion of water owing to the vaso-motor paralysis. There are many diseased conditions where, apparently, the same phenomena may occur ; thus in some instances a very highly concentrated but scanty urine may be secreted, and in others an abundant, highly dilute urine.

(3) *Synthetic activity.* — The renal substance is known to be

capable of synthesising hippuric acid from benzoic acid, and the latter when introduced into the circulation of animals is excreted in the form of hippuric. As mentioned above, it has been supposed that in the human kidney uric acid is synthesised. This view has been advanced amongst other reasons owing to the difficulty of demonstrating the presence of uric acid in the blood. Further, it has long been known that although birds secrete uric acid in abundance in the urine, their blood contains urea, and it has been suggested that the kidney of the bird is also the seat of formation of its uric acid. This view, at any rate as regards birds, is untenable, inasmuch as the destruction of the kidney of birds by injections of bichromate of potash is followed by the deposition of urates in the tissues; in fact the same effects are produced as those seen after ligature of both ureters in birds. In addition to this there is definite evidence that uric acid is synthesised in the liver of birds. For these reasons there must be considerable doubt as to the accuracy of the view that uric acid is formed in the human kidney.

(4) *Influence on metabolism.*—After the experimental removal of large amounts of the kidney substance, the excretory activity of the gland is maintained, but a disordered metabolism is produced, which, provided the quantity of kidney removed amounts to some three-quarters of the total kidney substance, leads rapidly to death. The removal of one kidney in man or animals is not followed by any appreciable disturbance either of the urinary secretion or of the general health; the remaining kidney undergoes a so-called hypertrophy, and many such patients have lived to advanced years. The removal of a portion of one kidney, amounting to from a sixth to a third of the weight of the organ, is followed in animals by considerable changes in the composition of the urine, although the general health of the animal remains unaffected. After such an operation the dog will excrete a more abundant urine than formerly, although the quantity of urea and other salts will not be affected. The increased excretion is usually temporary, although in some instances it has been prolonged in its duration. The removal of a sixth to a third of the weight of each kidney is followed by a permanent and great increase in the amount of urinary water; the general health of the animal remains good, and there is no increased excretion of nitrogenous extractives. If a portion of one kidney be removed together with the whole of the other, the increased excretion of urine is very marked and permanent, the quantity of urinary water being double, treble, or even quadruple the normal. The final result will depend on the quantity of kidney substance removed. If this amount to only two-thirds of the total kidney weight the animal will remain in good health for a prolonged period, *e.g.* eighteen months, although the urine will be dilute and greatly increased in amount. If the quantity removed amounts to three-quarters or more, a general rapid wasting accompanied by the excretion of large quantities of urea occurs,

and the animal dies in a condition of marasmus after an interval varying from one to six weeks. The marasmus and loss of flesh are not dependent on mere loss of appetite, as the animals will often eat considerable quantities of food. Notwithstanding the large excretion of urea the blood contains a great excess of urea and of other nitrogenous extractives, which would seem to point to the conclusion that the disorder of nutrition is dependent on an increased formation of urea, and not on a mere accumulation owing to any impaired activity of the renal excretion. It is of interest that the production of the marasmus and the subsequent death of the animal are dependent solely on the amount of kidney removed and not on the mode of its removal. The disturbance of nutrition does not ensue after the removal of portions of both kidneys, although it may be seen after the removal of a portion of one and the whole of the second. In the former condition a greater mutilation is effected than in the latter, but a smaller amount of renal tissue is removed.

These observations would tend to show that the physiological activity of the kidney in some way affects the metabolism of the nitrogenous tissues of the body, and that, therefore, the organ probably has other functions than those of mere excretion. There is, however, no evidence pointing definitely to the conclusion that the kidney has an internal excretion in any way analogous to those known in the case of the liver, the pancreas, the thyroid, and the suprarenal. Further, these observations would seem to throw some light on the increased excretion of urinary water seen in many diseases of the kidney, and also on the great loss of flesh that is associated with many chronic destructive diseases of the kidney. There was no evidence that the lesions of the kidney and the increased excretion of urinary water were in any way associated with any increase in the blood pressure, and no evidence of cardiac hypertrophy was found in any of the animals experimented on.

General Etiology of Renal Disease

Renal affections may be dependent on *anomalies*, congenital in origin, affecting the position or form of the organ, and described as malformations and displacements, or renal diseases may be acquired.

In the ACQUIRED DISEASES it is probable that in the great majority of instances the actual cause of the morbid process reaches the organ through its blood-vessels, either in the form of a *toxic substance*, or else as an infecting agent, *e.g.* a microbe, or as a macroscopic parasite-hydatid. This, however, is not invariable, and instances are known where the infective agent has reached the kidney through the

lymphatics or even by direct extension from adjacent parts. In other cases the origin of a morbid process is dependent on some alteration in the composition of the urine, as in lithiasis and the formation of calculi.

Cold.—In addition to the above factors in the production of renal disease it has long been supposed that there is an intimate relationship between the kidney and other parts of the body, more especially the skin, and that this relationship is effected through the nervous system; thus cold has been looked upon as a cause of several forms of renal disease, especially nephritis and Bright's disease, both acute and chronic. It was thought that the cold acting on the skin produced reflexly an intense congestion of the renal vessels through the activity of the vaso-motor system, and that this might be so severe as to lead to actual inflammation. The part played by cold in the production of disease is not now regarded as so direct as it formerly was, and in many affections of other parts of the body the action of cold is looked upon rather as a predisposing than as an exciting cause. The same is very probably true in the case of renal disease. There is no experimental evidence at the present time to show that there is any direct relationship between the activity of the skin and that of the kidneys other than the well-known fact that in cold weather more urine is passed, and in hot weather less. Stimulation of the skin by cold or other means does not, as far as is known, produce any direct effect on the renal circulation, and there is certainly no experimental evidence to justify the view that exposure of the surface of the body to cold causes any reflex vaso-motor effect leading to extreme congestion of the kidneys. It is possible that there may be some relationship between the secretion of sweat and that of the urine, but if such exists its mechanism at the present day is quite unknown, although it is true that in most forms of renal disease the secretory activity of the skin is greatly diminished; this, however, is not invariable, for patients suffering from acute or subacute Bright's disease may sweat copiously. Exposure to cold unquestionably favours the production of renal disease, thus these maladies are seen with great frequency in men working in hot shops, engineers, boiler-cleaners, etc., but in many of these it is difficult to exclude a toxic cause, such as alcohol or lead. Similarly cold will apparently produce renal disease during the convalescence or incidence of certain acute specifics, *e.g.* scarlet fever, syphilis, etc. Here also it is quite probable that the action of the cold is only a predisposing cause and not a direct exciting influence.

Passive congestion.—Among other conditions which may predis-

pose to the production of kidney affections is passive congestion of the organ. In valvular diseases of the heart, in certain lung diseases, and in abdominal diseases causing pressure effects on the vena cava or renal veins, there can be no doubt that passive congestion plays an important part in the production of chronic changes in the kidney. Long continued passive congestion will not only favour the production of nephritis, but it will also of itself lead to chronic changes in the organ which may seriously interfere with its efficiency as an excreting gland. In some instances, more especially in pregnancy, the occurrence of renal lesions has been put down to the mere effects of the mechanical interference with the circulation when it is more probable that the results are really of toxic origin. There is no definite evidence that the albuminuria of pregnancy is dependent on the action of a toxin, inasmuch as a toxic substance has not been isolated; but it is difficult, if not impossible, to regard the effects produced as dependent on the mere pressure of the uterine tumour. The fact that the albuminuria not uncommonly disappears as soon as the uterus is emptied, is equally capable of explanation on the hypothesis of a toxic cause as on that of a mechanical one.

In certain rare instances distinct effects are apparently produced on the circulation and secretion of the kidney as a result of *reflex irritation* arising in some other part of the body. It is well known that the passage of a catheter, especially in an elderly and broken-down patient, suffering from stricture, may occasionally be followed by complete suppression of urine and death from anuria. The same result is also occasionally seen as an accompaniment of acute diseases in other parts of the body; thus, for instance, perforation of an ulcer in the alimentary canal occurring where there has not been previously any marked ill-health, may be followed by sudden and complete suppression of urine, which is rapidly fatal. The same phenomenon has been seen in certain instances of disease of the bladder without the previous existence of any serious renal disease. It has been supposed that these facts must receive their interpretation on the hypothesis that the changes produced in the kidney, circulatory or secretory, are brought about through the nervous system. In some instances quoted this would seem possible, but in others it is again extremely difficult to eliminate a toxic or chemical cause.

Toxic substances.—The action of toxic substances may be looked upon as the cause of so-called acute and chronic inflammatory diseases of the kidney, and doubtless also of certain

degenerations of a chronic character, such as granular kidney, lardaceous disease, etc.

The toxic substances capable of producing morbid processes in the kidney may be in the form of poisons introduced with the food, or possibly they may be substances liberated by disordered processes of metabolism in the body, or, finally, they may be substances elaborated by the activity of micro-organisms causing morbid processes in other parts of the body than the kidneys.

It has long been recognised that certain affections of the kidney, as for instance some forms of Bright's disease, have in many instances a toxic origin, but it is only recently that the view has gained ground that all forms of so-called nephritis, and probably all forms of Bright's disease, are really of toxic origin. Not only are there a great variety of substances which can be shown to have a toxic action on the kidney, but there is frequently such a differentiation in the action of these toxic substances as to render them capable of being classified more or less roughly according to their action. Many writers consider that two groups of toxic substances can be differentiated according as the main action is exercised on the glomeruli or on the renal tubules. Others include a third group, the members of which have a somewhat different action. In the first group the renal poisons acting mainly on the glomeruli and producing glomerular nephritis are included. Cantharides affords an instance of such a poison, and it is probable that the action of the virus of scarlet fever produces closely allied results. The heavy metals, especially lead, afford a typical instance of the renal poisons of the second group, the main action of these being to produce a coagulating necrosis of the cells of the convoluted tubules. The salts and the oxides of the heavy metals are especially prone to produce this effect, and very similar phenomena are seen as a result of the action of chromic acid and the bichromates. The third group of renal poisons contains a number of complex animal and vegetable substances having a toxic action on the renal structures. The best known instances of these poisons are snake venom, abrin, ricin, and the blood of certain fish, especially that of the eel. These poisons produce a degeneration of the tubules of the kidney characterised especially by vacuolation and degeneration of the renal cells. The effects produced on the renal epithelium are somewhat different to those seen after the action of heavy metals, owing to the fact that in the former case the nuclei of the cells persist, whereas in the latter the coagulating process is so complete as to destroy the entire cell. One of the most remarkable renal poisons hitherto

discovered is the substance vinylamine, a primary amine of vinylic alcohol. This substance is not only capable of producing in certain doses a so-called acute nephritis, but in smaller doses administered for prolonged periods it produces more chronic lesions. Vinylamine produces an acute congestion of the blood-vessels, the glomeruli being dilated and engorged with blood. The epithelium of the tubules becomes vacuolated and the cells become irregular in shape; the nuclei, however, persist. Lindemann, who has described the action of this substance, is of opinion that the protoplasm of the renal cells undergoes a progressive solution, and that portions of it pass into the stream of urine. The formation of casts is very abundant, especially where the toxic action is prolonged. The changes produced by this substance are limited to those in the glomeruli and in the convoluted tubules, the straight tubules not being affected, and it is of interest that no interstitial changes are produced even by the chronic action of the poison. Vinylamine produces renal lesions very similar to those produced by toxins of microbic origin, and also to those produced by animal and vegetable proteid poisons. Lindemann considers that somewhat similar results can be seen to follow the action of the bodies cadaverin and putrescin. All these substances produce marked effects on the renal structures, but there are other means of producing slighter changes in the renal tissues. It has long been known that the injection of the blood of one animal into another is liable to cause slight changes in the kidneys together with the production of albuminuria. In the case of the rabbit, for instance, the most toxic serums are those of the cat and the guinea-pig. Lindemann has described experiments tending to prove that the serum of the guinea-pig may be made to acquire very considerable toxic properties on the kidney of the rabbit by injecting the guinea-pig with the emulsion of rabbit's kidney. If the serum of a guinea-pig that has received injections of an emulsion of rabbit's kidney be injected into a rabbit, albuminuria is produced within a few hours, and death from complete suppression of urine may occur in twenty-four hours. These experiments show at any rate that the blood of animals may contain minute quantities of substances capable of producing very profound renal changes, and that therefore our view must be enlarged as to the nature and variety of renal poisons capable of producing effects in the kidney resembling those seen in morbid processes.

Alcohol and lead are the two most important toxic substances producing renal disease introduced with the food, and in both cases the renal disease produced by their action is most likely

to be subacute or chronic. The action of the two poisons is very often combined in the same patient. Lead is capable of producing serious and fatal renal disease, chronic in its form, without of necessity ever having given rise to lead colic or the more acute manifestations of plumbism (Vol. II. pp. 52 and 65).

Our knowledge is far less definite as regards the toxic substances causing renal disease produced by erroneous metabolism. Gout, however, may be quoted as a malady in which there is some anomalous metabolism, and where it is possible that the renal changes are produced by this condition, although renal disease is by no means an invariable accompaniment of gout. Some writers have supposed that lead produces the renal lesions indirectly owing to its first producing gout. The fact that complicated organic substances, like cadaverin and putrescin, are capable of producing lesions makes it by no means improbable that the by-products of an abnormal metabolism may be looked upon as one of the causes of the production of renal disease. There is at least one renal lesion, the granular kidney, which is held by many authorities to be associated with so-called high living.

The urine serves as a channel of excretion not only for the toxic substances elaborated by micro-organisms, but in many conditions the organisms themselves are also excreted, though it is probable that this does not occur until the efficiency of the kidney as a filter has been impaired. Micro-organisms when injected into the blood stream do not reappear in the urine of healthy animals, but this may be in part dependent on the fact that they are so rapidly removed from the blood. Micro-organisms not only appear in the urine in diseases involving the urinary structures, but also in cases of general infection. Thus the bacillus tuberculosis may be found in the urine and free in the renal tubules in cases of acute tuberculosis, and the bacillus of typhoid fever is almost constantly present in the urine during the height of the disease. In both these diseases the specific microbes may be present in the urine notwithstanding the fact that the kidney has not undergone any grave structural alterations, but its integrity as a filter has been impaired, as is shown not only by the presence of the organisms, but also by that of small quantities of albumin.

The products of the activity of micro-organisms are also excreted in the urine, as is well known in the cases of the bacillus pyocyaneus, and sometimes they are present in considerable abundance, as, for example, in the albumosuria so constantly seen in pneumonia, empyema, etc.

Organisms producing disease primarily in some other part of the body may cause renal disease, thus renal tuberculosis may be set up by the arrest in the kidney glomeruli of the bacilli circulating in the blood, and derived perhaps originally from some caseous focus in a distant part of the body. Multiple miliary abscesses may result in a similar way from the arrest of staphylococci and other pyogenic organisms, producing actual infarcts in the kidney. It would seem that in some instances chronic renal disease similar to the granular kidney, and of the type of interstitial nephritis, may be started in a similar manner.

It is more frequent for renal disease to be produced by the action of a toxin, and this is notably the case in such a disease as diphtheria, and possibly pneumonia. Scarlet fever is, however, of all the acute diseases the one most liable to be followed by inflammatory renal affections, such as the various forms of nephritis and Bright's disease. The nephritis of scarlet fever is in all probability of toxic origin, although the nature of the toxic substance present in the blood is unknown. There is a great similarity between the lesions produced by the virus of scarlet fever and those seen as a result of the action of cantharides. Nephritis may be seen during the course or convalescence of any acute febrile disease, and more especially of the acute specific diseases; typhoid fever, pneumonia, diphtheria, measles, rheumatism, syphilis, are, however, all of them liable to be followed by somewhat similar effects.

The classification of the various morbid processes started in the kidney as a result of these acute specific diseases is unsatisfactory, but it may be stated broadly that at least two effects may be produced. The first is seen during the height of the initial acute specific disease, and the second is more frequently seen as a complication occurring during the subsidence of the original malady or during convalescence. The renal complication which occurs during the height of the initial disease is best described as a transitory nephritis, and is described by English writers as a congestion of the kidney characterised clinically by the presence of albumin in the urine, for which reason the whole condition has often been spoken of as one of "febrile albuminuria." Though this transitory nephritis is seen during the height of the initial malady it does not accompany it from the onset; thus in diphtheria the albuminuria is not usually marked until the third or fourth day, and even in scarlet fever and pneumonia it is not necessarily present from the beginning. The condition disappears with the subsidence of the original attack, and the urine regains its normal characters notwithstanding the fact that

the amount of albumin present may have been considerable. In this transitory nephritis albuminuria is the most marked departure from health, traces of blood may be present, but anasarca and other symptoms associated with graver disease of the kidney substance are not usually present. This febrile albuminuria is unquestionably of toxic origin, and dependent on the action on the kidney substance of the toxins or microbes producing the primary disease.

The more serious complications characterised by the terms acute nephritis, progressive nephritis, Bright's disease, occur as a rule during the later stage of the original primary malady. It is remarkable that in such a disease as scarlet fever or pneumonia, an albuminuria dependent on a transitory nephritis may occur during the height of the disease and subside completely, to be followed during convalescence by a further albuminuria associated with other grave symptoms, and dependent on a much more serious lesion of the kidney, to which the term Bright's disease may be applied. It is possible that these two renal lesions having a somewhat similar etiology may be really more closely allied, and that one is a graver form of the other. It is quite certain, however, that they may, as stated above, occur at different times in the course of the original disease, and that the one does not necessarily pass into the other. It is highly probable that the more serious lesion is also produced by toxic substances present in the blood and in the urine, although for many years it was a favourite doctrine that the onset of Bright's disease during convalescence from an acute specific was due to the action of cold. Striking instances of this connection are certainly sometimes seen in cases of scarlet fever and in cases of secondary syphilis, where exposure to cold and wet has apparently been followed by the development of acute or chronic Bright's disease. Some of the most acute forms of Bright's disease, however, may be seen in cases of scarlet fever where every possible precaution has been taken to prevent the action of cold. In the opinion of the writer the action of cold, in causing such a malady as Bright's disease, is probably an indirect one, just as it is in the production of such an acute specific disease as pneumonia. It is quite possible that cold may act in such a manner as to lower the resistance of the tissues, the kidneys amongst others, and in that way favour the action of toxic substances on the renal structures.

Pregnancy is also associated in some way with the production of nephritis and of Bright's disease, and here also it is probable that the active agent is a toxic one, although its identity has not been established. The albuminuria of pregnancy resembles that

seen as a result of acute specific diseases, and all varieties are found between a simple albuminuria like that dependent on what has been described as a transitory nephritis partial in its distribution, and the most severe form of Bright's disease involving all the renal structures. The renal complications of pregnancy may not only clear up after delivery—although an interval of some weeks may elapse before the complete disappearance of the albuminuria—but they may return with subsequent pregnancies, though this is not invariably the case.

Whilst the tendency is to regard all forms of nephritis and Bright's disease as primarily of toxic origin, there are many *predisposing causes* favouring the action of the toxic bodies of which the most important are the age, occupation, and habits of the patient. The more severe forms of Bright's disease are especially prevalent in young adults, although the malady may be seen both in children and in middle-aged persons. The more chronic forms of renal disease, especially those of a degenerative type, such as granular kidney, occur in the more elderly and are more especially prevalent in patients from forty to fifty years of age.

Sex.—Renal disease is more common in men than in women, probably owing to the fact that the former are more liable to be exposed to the action of such toxic agencies as lead, alcohol, and syphilis.

Occupation plays a great part in the production of renal disease, not only on account of the vicissitudes of temperature to which certain workmen may be exposed, but also owing to the large number of occupations in which lead may enter as a possible causative agent—painters, plumbers, engineers, fitters, etc. It is also difficult to exclude the action of alcohol in many of these instances, and it is very probable that the combined action of alcohol and lead is peculiarly prone to be followed by the development of renal disease.

General Morbid Anatomy and Pathology of the Kidneys

Anomalies.—The kidneys may be anomalous in their position, their form, or their number, but very frequently these departures from the normal are combined, so that the organ, anomalous in position, is also of abnormal shape. A third or supernumerary kidney has been observed in rare instances, but is by no means common, as in the case of the spleen and adrenals. The presence of a single kidney may be due to the want of development of its fellow, to the atrophy

of one as a result of disease occurring in fœtal life, or to the fusion of two primarily distinct kidneys. The want of development of one kidney is shown not only by the absence of any remains of kidney substance, but also by the complete absence of all trace of the renal vessels and the ureter. Where atrophy of one kidney has occurred in fœtal life some trace of the renal vessels and of the ureter can usually be found even when the kidney fragment is extremely small. The causes of atrophy of one kidney in early life are obscure, but it is at any rate certain that it may be associated with obstruction of the renal artery, obstruction of the ureter, and, in some cases, with some want of development or anomaly in the mode of development of the kidney itself. This may be shown by the fact that, although the kidney fragment may be of some size, yet it is anomalous in its structure, so that, for instance, it may contain no glomeruli. A single kidney associated with complete want of development or atrophy of its fellow is not only much larger than the normal kidney, but frequently weighs more than the sum of the weights of the two normal kidneys. Instances are on record where a single kidney has weighed as much as a pound or more.

The commonest form of single kidney resulting from the fusion of two originally separate organs is the horse-shoe kidney, the lower ends of the organ being united by a band of renal tissue which crosses the vertebral column opposite the second and third lumbar vertebræ. In some instances the band of union passes between the upper extremities of the kidneys, and in others it consists of fibrous tissue only, containing no renal elements. A horse-shoe kidney is usually of irregular shape, and the lobulation is well marked. It is supplied by several renal arteries, and the two or more ureters leaving it usually pass over the front of the mass.

In other instances of single fused kidney the two organs, instead of being joined by a band of renal tissue, are fused end to end, or else are connected by their surfaces becoming adherent. In both these latter conditions the two ureters are found on the same side of the body, but they open in their normal positions into the urinary bladder.

Anomalies in Position.—The kidneys may not only be unnaturally movable in the retro-peritoneal tissue, but they may occupy quite abnormal situations. The most common of these are displacements downwards over the sacro-iliac synchondrosis or into the iliac fossa, or, in rare instances, into the concavity of the sacrum. Such displacements of the kidney are often associated with malformations of the large intestine, and in some instances displacement of the kidney

into the concavity of the sacrum is accompanied by the condition of imperforate anus.

The ureters often present abnormalities, the most frequent being that for a variable distance one ureter is double, in other words, the calices are unduly prolonged. A complete double ureter possessing independent orifices into the urinary bladder is very rare. The renal pelvis may be abnormal, so that it is large and pyriform, the dilatation ending suddenly where the ureter commences. In other instances the ureter may be abnormally narrow, or may be implanted at an acute angle into the renal pelvis, and under these circumstances the abnormality may be a cause of hydronephrosis. Occasionally both ureters and also the renal pelvis are greatly dilated, and this condition, if of congenital origin, is not invariably associated with the presence of an obstruction in the more distal part of the urinary tract.

Movable kidney.—Two varieties of movable kidney are generally recognised, differing, however, only in the degree of mobility present. In the less severe form the kidney is more or less freely movable in the retro-peritoneal tissue. In the more movable variety, sometimes termed "floating kidney," a partial mesonephron may be present, either of congenital origin, or perhaps more frequently it has been gradually formed as a result of the unnatural mobility of the organ.

The excessive mobility is associated with a number of other anatomical changes, such as laxity of the abdominal walls, disappearance or want of development of the renal fat, and changes in the reflections of peritoneum passing from the anterior surface of the kidney to the abdominal parietes and neighbouring viscera. The right kidney is more often affected than the left, and when the affection is bilateral it is often more developed on the right side. In some instances the mobility of the kidney leads to kinking of the ureter, with the production of hydronephrosis, or to the kinking of the renal vein and the development of passive congestion of the organ of varying extent. Dilatation of the stomach is not an uncommon accompaniment of movable kidney, and is regarded as dependent on the traction exerted on the duodenum and pylorus through the peritoneal reflection passing from the kidney to these organs. In a similar way the bile duct may be obstructed.

Congestion of the kidney.—The most extreme forms of passive congestion of the kidney are seen in cases of thrombosis of the renal vein occurring during the course of malignant disease of the kidney or of pyelo-nephritis. In other instances the thrombosis

may occur as a complication of granular kidney and as an accompaniment of clotting commencing in a pelvic vein, and gradually spreading to and involving the vena cava. Thrombosis of the renal vein is also rarely seen in marasmus and as a complication occurring in convalescence from acute disease where great debility has occurred, *e.g.* diphtheria. The more common but less severe varieties of passive congestion are seen as a result of heart disease and such lung diseases as bronchitis and emphysema, where the return of the blood through the venous system is hindered. All diseases of the abdomen or mediastina causing obstruction to the circulation through the vena cava will necessarily produce varying degrees of passive congestion of the kidney, *e.g.* mediastinal growths, adherent pericardium, cirrhosis of the liver, and certain abdominal tumours.

The anatomical appearance of congested kidneys varies according to the severity and the duration of the passive congestion. Where the renal vein is completely blocked by a thrombus the congestion is extreme, the vessels of the medullary portion of the kidney being gorged with blood, and the organ greatly increased in size. In extreme cases the congestion affects not only the medullary portion of the kidney, but also the mucous membrane of the pelvis, and extravasations of varying size may be found beneath this mucous membrane, and both in the interstitial tissue between the straight tubules and in the interior of the straight tubules themselves. The congestion involves also the cortex of the kidney, and the glomeruli not only have their vessels greatly distended but the glomerular chamber may contain extravasated blood; the distension of the Malpighian bodies is, however, not usually so great as that seen in active congestion. The stellate veins on the surface of the organ are also engorged, and in cases where the passive congestion has existed for some time the organ may be unduly adherent to its fatty capsule.

Overgrowth of fibrous tissue occurs in cases where the passive congestion has existed for a considerable period. The increase of connective tissue is especially marked in the interstitial tissue between the tubules and around Bowman's capsule. The basement membrane of the tubules also undergoes thickening. The cells lining the convoluted tubules of the kidney have frequently lost their characteristic striated appearance, and become cubical in shape and hyaline in structure, and similar changes may involve the cells of the straight tubules.

EMBOLISM AND THROMBOSIS OF THE RENAL VESSELS.—Occlusion

of the renal artery or its branches by emboli is very common, thrombosis is rare.

The anatomical fact of the existence of end arteries in the kidney greatly favours the production of embolism. An obstruction of even small branches of the renal arteries is necessarily accompanied by the necrosis of a larger or smaller area of the cortex of the kidney. The necrosis only involves the medullary portion of the organ when a very large branch of the renal artery has been occluded, it would seem as if the collateral anastomoses in the medulla are sufficiently free to maintain the circulation when the occlusion affects vessels of moderate size only.

The infarctions produced by embolism vary a good deal in appearance. When small they are very often wedge-shaped with the base of the wedge at the surface of the kidney, when larger they are not uncommonly quadrangular in section, and when the occluded vessel is quite large the resulting infarct is often of most irregular shape. The further changes and appearances of the infarct depend a great deal on the nature of the embolism, whether it be of septic origin or not. In cases of simple embolism similar results are produced to those seen following ligature of a branch of the renal artery. The area of kidney involved undergoes necrosis and assumes a deep yellow colour, owing to the degenerative changes occurring in the portion of kidney where the circulation is arrested. The yellow necrotic area is separated from the healthy renal substance by a zone of congestion which is dependent on the back-flow of blood from the veins of the surrounding healthy area into those of the region where the circulation has been blocked. In the very early stages of the formation of an infarct the whole area, which will be subsequently yellow in appearance, may be of a deep red colour owing to this back-flow, and the engorged area may be raised slightly above that of the surrounding parts. The degenerative changes accompanying the necrosis occur rapidly, and the yellow colour may appear in two or three days and may persist for many weeks. Subsequently the necrosed region undergoes absorption, and its site is marked by a puckered scar.

If the embolus contain infective material, suppuration of the necrotic area may take place, and in such instances the inflammatory reaction round the infarct is very considerably greater in amount than in the cases of simple embolism. Septic infarcts may be associated with inflammatory changes, not only in the kidney, but also in the capsule and in the perinephritic fat.

Thrombosis of the renal artery or of its branches is a rare

lesion notwithstanding the frequent occurrence of disease of the inner coat of the vessel, which may be half occluded from this cause. Thrombosis of the interlobular and glomerular arteries occasionally occurs, and blocking of the glomerular arteries is often associated with hyaline degeneration of the vessels of the glomerular tuft. Widespread thrombosis of the interlobular arteries has been described producing anatomical changes in the kidney, essentially similar to those seen as a result of infarction. The main interest of the condition lies in the fact that, although the secretion of urine in such a case is necessarily completely arrested, the clinical symptoms produced are similar to those seen in calculous anuria.

ACTIVE CONGESTION OF THE KIDNEY.—It is difficult, if not impossible, to separate active congestion from NEPHRITIS, inasmuch as nephritis is accompanied by extreme congestion, and it is only in the earliest stages that the morbid changes are limited to the blood-vessels. In the more severe forms of acute congestion the kidneys are swollen, and their volume may be increased to twice that of the normal; in some instances the capsule may even be split. The distension of the blood-vessels is especially marked in the cortex, and the glomeruli may often be seen as bright red glistening points. The vascular distension may involve also the medulla, and the red appearance of the section of the kidney may be in part due to the occurrence of hæmorrhage into the renal tubules and also into the interstitial tissue.

In some forms of acute nephritis, especially when of septic origin, the vascular engorgement is not uniformly marked all over the cortex, the hæmorrhages being distributed in a more or less punctiform manner. This is very obvious on the surface of the kidney. The cortical substance, especially if the nephritis be of some duration, may be of a pale yellowish colour, owing to the necrotic changes in the epithelium following as a result of the vascular lesions.

Microscopically the vessels of the glomerular tuft are greatly distended, and Bowman's chamber may be filled with blood corpuscles that have escaped from the vessels. In the slighter forms the extravasation of blood corpuscles is less marked, and the chamber is filled with an albuminous exudate. The glomerular tuft and the lining membrane of Bowman's capsule may both show signs of proliferation if the nephritis be severe or of long duration. The epithelium of the convoluted tubules loses its ordinary appearance as the cells undergo cloudy swelling, and in the more severe forms of congestion and

nephritis necrotic changes may be marked and the cells lining certain portions of the tubules may be shed, the tubules themselves containing casts which are frequently formed of coagulated blood. The interstitial tissue is swollen and œdematous, the blood-vessels engorged, here and there hæmorrhages may be seen between the tubules, and where the condition has lasted for some time the early stages of fibrous overgrowth may be present, together with marked leucocytic infiltration.

The lesions produced by acute congestion and nephritis are not usually uniformly distributed throughout the kidney substance, being more marked in some places than in others. In the nephritis following the acute specifics the leucocytic infiltration may be disseminated in such a manner as to produce scattered accumulations of leucocytes of sufficient size to be visible to the naked eye. The name of "lymphomatous nephritis" has been given to this variety. Glomerular changes and tubular changes are not usually equally marked in any given case of nephritis; in some the former are more advanced, in other cases the tubular; in others the lesions in the interstitial tissue reach a higher degree of development than those affecting the kidney elements themselves, so that a glomerular, a tubular, and an interstitial nephritis may be recognised. In glomerular nephritis the lesions are well marked in the glomerular chamber and tuft, but some changes will be found in the tubular epithelium and some in the interstitial tissue, so that the distinction is rather one of degree than of kind. Owing to the vascular arrangements of the kidney it is impossible for the lesion to advance to a high degree of development in the glomeruli without affecting to some extent the epithelium of the tubules.

Bright's disease.—The term Bright's disease is used by some as including many forms of nephritis, and by others as descriptive of more chronic lesions of the kidney, sometimes regarded as the sequel of inflammatory changes. Many writers consider acute Bright's disease and acute nephritis as synonymous, and certainly it is difficult to draw any hard and fast line of distinction between the two as regards their morbid anatomy. Clinically one of the great distinctions between acute Bright's disease and acute nephritis lies in the fact that the former is generally accompanied by dropsy and the latter not, but still the occurrence of dropsy in acute Bright's disease is not invariable. The difference amongst writers is largely a question of the use of terms, as there is a very real distinction between the acute nephritis seen, for instance, as an accompaniment of acute febrile disorders, and the acute Bright's disease which may

result either during the convalescence from these acute febrile diseases or from other causes.

Acute Bright's disease is not only a very much more serious affection both as regards the lesion produced in the kidney and as regards its clinical symptoms, but it also has no direct relationship with the nephritis arising during the height of infective diseases causing it. Thus scarlet fever may give rise to a transitory nephritis acute in its onset but mild in its course. This accompaniment occurs during the height of the fever, and with the subsidence of the pyrexia it also diminishes. But scarlet fever may also give rise to acute Bright's disease, either during the height of the disease or more commonly during convalescence. It is, of course, possible to look upon both these renal complications as nephritis, simply regarding the one as a more serious form than the other, and on this view acute Bright's disease would be a more severe form of acute nephritis, and the term Bright's disease would remain limited to the chronic form of the malady.

In the early stages of acute Bright's disease the kidneys are swollen and distended with blood, this gives the organ a dark red colour. The congestion is especially marked in the stellate veins of the cortex. The capsule strips off readily, leaving a smooth surface, the colour of which depends on the duration of the disease. In the earlier stages it may be of a uniform red colour and congested, in the later stages it may be uniformly white or wax-like in its appearance, and in the intermediate stages it is marbled. The cortex may assume a uniform wax-like appearance in as short a time as six weeks from the onset of the malady. On section the cortex is found to be increased considerably in width. The glomeruli can often be seen standing out as bright red glistening points, and streaks of hæmorrhage may be seen between the tubules both in the cortex and in the medulla. The medulla is usually deeply congested and of a dark red colour, the contrast between the yellow opaque cortex and the congested medulla being very marked. Bowman's chamber is usually filled with fibrinous exudation and extravasated blood corpuscles. The products of inflammatory exudation may be found between the loops of the capillary tufts as well as free in the glomerular chamber. Thrombosis may occur in the vessels of the capillary tuft, and hyaline degeneration affecting their walls is also common. The epithelium lining Bowman's chamber may undergo proliferation, and the mass so formed may be sufficiently large to compress the capillary tuft. The loose connective tissue outside the glomerular chamber may also show signs

of exudation and proliferation, and it is owing to this that in the later stages of the disease the glomerular chamber undergoes thickening. All the changes affecting the glomeruli are of a character to interfere with the circulation through it, and it is doubtless in part owing to these lesions that the epithelium of the convoluted tubules undergoes change. The cells of the convoluted tubules lose their characteristic striated appearance, become hyaline, and often undergo fatty degeneration. In the most severe forms of acute Bright's disease the cells lining these tubules may be shed *en masse*, large tracts of the tubules being destitute of their cellular lining. The tubules become dilated, and the lumen is occupied by masses of disintegrated cells, exudation, and blood corpuscles. It is owing to the obstruction to the flow of urine produced by these disintegrated renal elements that the tubules undergo dilatation, and this in part accounts for the increased size of the organ. The changes in the medulla are of a similar character to those present in the cortex, but they are usually less marked in amount, although hæmorrhages between the tubules are by no means uncommon, and the cells lining the tubules may often become hyaline and glass-like. The interstitial tissue is œdematous, and in the more severe forms shows the presence of inflammatory exudation and leucocytic infiltration, although the extent of this is very varying in different cases of Bright's disease. These changes are generally well marked in the vicinity of the small arteries, producing varying degrees of peri-arteritis, and if the malady have lasted a few weeks end-arteritis may also be present and cause very considerable narrowing of the lumen of the vessels. The swelling of the interstitial tissue is also partly responsible for the increase in the size of the organ.

Chronic Bright's disease.—A classification of the different forms of chronic Bright's disease based on the morbid anatomy exclusively is unsatisfactory, but, speaking broadly, two varieties, distinct both in their anatomy and to a great extent in their clinical course, may be recognised.

In the first the so-called *large white kidney*, one of the leading features, is a considerable increase in the size of the organ, and it is from this as well as from its colour that it has derived its name. A great increase in the size of the organ is not seen so often now as formerly, and this is owing to the fact that lardaceous degeneration is not so common as it was. This change is often present to a very variable extent in the kidneys of chronic Bright's disease, and in former years many specimens described as cases of

chronic Bright's disease with large white kidneys were really instances of lardaceous infiltration. The large white kidney may weigh from six ounces to one pound, but instances of such extreme enlargement are rare, and the increase in size and weight is usually only moderate. The surface of the organ is pale and smooth, in some instances it is marbled. The capsule is not usually much thickened, and on stripping leaves a smooth surface on which the stellate veins are extraordinarily distinct. On section the cortex is thickened, opaque, yellowish white in appearance, and the contrast between the pale cortex and the congested pyramids is very marked. All the elements of the kidney are affected by the morbid process. The glomeruli are generally increased in size, and the Malpighian capsule is thickened. The epithelium lining the glomerular chamber is proliferated, and the glomerular tufts present evidence of cellular infiltration. The capillaries undergo hyaline degeneration, and Bowman's chamber is often partially filled by coagulable exudation. The changes in the epithelium of the tubules vary in different parts of the kidney. The tubules, speaking generally, are dilated and in places the epithelial lining is shed. In others the cells have undergone hyaline or fatty degeneration, and the lumen of the tubules is occupied by casts. In places the renal tubules may present a fairly normal appearance, the incidence of the morbid process not being usually uniform. The interstitial connective tissue undergoes overgrowth, and the formation of fibrous tissue may be considerable, although slighter in amount than that seen in the other variety of chronic Bright's disease. Delicate in its structure, one of the most important characteristics is the uniform degree of its development throughout the kidney.

In both varieties of chronic Bright's disease arterial changes, peri-arteritis and end-arteritis, are well marked in the renal vessels.

In the second variety of chronic Bright's disease the kidneys are very considerably diminished in size, their weight falling to three or even to two ounces. The capsule is thickened, and on stripping leaves a roughly granular surface, which often differs from that seen in the true granular kidney, in that the capsule in chronic Bright's disease is not so adherent to the kidney substance as in the other affection, and the granules in the granular kidney are often much smaller than those seen in the second variety of Bright's disease, to which the name of *contracted kidney* or *small white kidney*[1] may be given. The colour of the cortex varies a good

[1] It is difficult to give a suitable name to this affection. The term "small white kidney" is sometimes applied to it, but is also used for another and rarer condition in

deal, in some cases it may be yellowish white or greyish in appearance, in others it is more mottled, but the distinction between the cortex and the medulla is always well marked, and the former is greatly diminished in width and may measure not more than one-sixteenth of an inch. The morbid changes are developed to different extents in different parts of the kidney, but the glomeruli generally are diminished in size and their capsules are greatly thickened, and as a result of this and of fibrous overgrowth in the glomerular tuft the latter is compressed and shrivelled; generally the glomeruli do not show signs of epithelial proliferation. The most characteristic feature as regards the tubules is the loss of their epithelium, and throughout extensive areas of the kidney the convoluted tubules may simply remain as collapsed tubes destitute of epithelial lining. Scattered areas may be found where the tubules are still lined by epithelium, not greatly differing in characters from the normal. In other parts the epithelium has undergone hyaline degeneration, and in many places certain tubules may have undergone considerable dilatation, so as to form actual cysts, microscopic in size or even visible to the naked eye. The interstitial tissue is greatly increased in amount, and in places the overgrowth is so considerable that nothing but masses of fibrous tissue in which a few atrophied glomeruli and shrunken tubules are to be found.

LARDACEOUS KIDNEY.—The etiology of lardaceous degeneration of the kidney is the same as that of the lardaceous disease of the body generally; the various forms of tuberculosis, suppuration in bone, and syphilis are its most frequent causes. In many diseases, for instance in phthisis, the kidney may not only present the characteristic changes, but in addition those of chronic Bright's disease, and it is by no means uncommon to find the lesions of Bright's disease present in the epithelial structures of the organ, and those of lardaceous disease in the blood-vessels and glomeruli. The kidney is not affected by waxy degeneration in the course of the diseases causing it as early as the alimentary canal, but marked changes may occur in the kidney without the liver and spleen being affected to a sufficient degree to cause obvious physical signs.

In lardaceous degeneration of the kidney the organ is usually increased in size, but in a few instances the change has been present to a

which the kidney presents many of the appearances seen in the large white kidney, but is of small size and its surface is not granular. The term "contracted kidney" is sometimes applied to the granular kidney, a totally distinct affection. It might be better to use the word atrophic to describe the second variety of Bright's disease, and the word hypertrophic for that usually known as the large white kidney.

marked extent in shrunken and granular kidneys. On stripping the capsule, which is seldom thickened, the surface of the kidney is smooth, and on section the cortex presents a great contrast in its opaque yellowish colour to the pyramids, which are commonly deeply congested. In the earlier stages the lardaceous degeneration affects mainly the blood-vessels, and especially those of the Malpighian tufts. The alteration is said to begin in these and to affect the vessels of the medulla subsequently. Where the degeneration is more marked the tubular structures are also involved, especially the basement membrane. The epithelium of the convoluted tubules not only undergoes lardaceous change, but in marked instances of this disease the cells may also become granular and fatty.

Granular kidney. — Granular kidney is usually classified amongst the degenerations of the organ, although some writers still include it among the inflammatory affections as one of the possible varieties of Bright's disease. The true granular kidney has no relation to Bright's disease in the strict sense of the term, although in one variety of Bright's disease extreme cirrhosis of the kidney may occur, producing a superficial resemblance both in the clinical symptoms and in the morbid anatomy to the conditions seen in the true granular kidney. Senility, and especially premature senility, are important factors in the etiology of granular kidney. Although especially prevalent in middle life it may be seen almost at any age, and a number of cases have been described even in young children. Where it occurs in children and in young adults hereditary or family tendencies to the disease may in some instances be traced, and there are several recorded instances where several children of one generation have been affected by this malady. The disease is more common in men than in women, in the proportion of at least two to one.

If the cirrhotic or contracted form of Bright's disease be excluded there are still remaining at least two varieties of granular kidney. In one the renal lesion advances to a high degree of development, and the general arterial lesions, although present, are not necessarily very advanced. In the other the general arterial lesions are widely spread and are of an advanced character, whereas the anatomical changes in the kidney may be, comparatively speaking, slight. These facts are of importance in regard to the etiology of the disease, as in the cases where the arterial lesions are widespread and the renal lesions comparatively slight the etiology of the affection is really that of general arterial sclerosis. Amongst the factors causing this may be included age, sex, occupation, especially

one involving strain, high living, and long-continued worry and mental anxiety. In addition to these it is very probable that such lesions may develop as a result of toxic agencies, and there would seem to be but little doubt that lead, at any rate amongst mineral poisons, is capable of producing not only arterial lesions but also granular kidney. The influence of alcohol has been variously estimated by different writers. According to some it is a potent agent in the production of granular kidney, others think that it is more likely to produce one or other of the forms of chronic Bright's disease, but there is a general consensus of opinion that arterial degeneration and granular kidney may certainly supervene as a result of high living, and some writers consider that the malady may be brought about from an excessive meat diet.

Granular kidney is not uncommonly associated with gout, but by no means invariably, and in some instances where the two diseases are combined lead has played a part in the etiology of both. Still gout is looked upon by many as a direct cause of granular kidney.

Arterial degeneration and granular kidney are especially prevalent in patients suffering from various forms of chronic insanity, and they may be present as complications in other diseases, *e.g.* myxœdema; and in reference to this point it is of interest that the removal of the thyroid in dogs is followed by the development of albuminuria.

At least two forms of granular kidney must be recognised apart from the atrophic or contracted white kidney, which has been described as a variety of Bright's disease. In one form of the true granular kidney the renal lesions are but the accompaniment of a widespread arterial degeneration, and are not much more developed in the kidneys than in other parts of the body. In the second variety the changes in the kidney have advanced to a very much greater extent than those in the arterial system generally, and it is probable that the renal lesion is primary.

The size and appearance of the organ vary considerably in these two different varieties. In the variety affecting the general arterial system the kidneys may be but little below the normal size, whereas in the other form they are greatly diminished in bulk and may weigh but two ounces a-piece or even less. In both forms the capsule is unduly adherent, and on stripping tears the renal substance, leaving a granular and reddish surface, the size and prominence of the granules depending greatly on the degree to which the kidneys have shrunk. The surface of the organ may

be deeply congested or mottled, but in some cases it is pale, and under these circumstances such a kidney approaches in its characters to that described as the contracted form of Bright's disease. On section the cortex is seen to be greatly diminished in amount, and frequently measures not more than one-sixteenth of an inch. The cross sections of the vessels stand out prominently, the medulla is congested, and owing to the great diminution in the amount of the renal substance the fat and connective tissue in the hilum seem to be unduly abundant. Cysts, from the size of a pin's head to that of a cherry, are frequently seen scattered in the cortex of the organ, and in some instances the formation of cysts is abundant. There is usually an enormous overgrowth of fibrous tissue in the kidney, but this, although widespread, is not uniform in its distribution. Areas of varying size, consisting of little but dense fibrous tissue containing the remains of degenerate fibroid glomeruli, are separated from one another by smaller areas containing dilated tubules and glomeruli which, although thickened and fibroid, are not completely destroyed. The fibrous overgrowth is most marked round the glomeruli, and Bowman's capsule is greatly thickened, and the glomerular chamber correspondingly diminished in size. The connective tissue is also present in abundance in the capillary tuft, and in those portions of the kidney where the glomeruli are comparatively slightly affected these frequently undergo hyaline degeneration. There is widespread destruction of the tubules, especially in the cortex, and over large areas the basement membranes are their only remains. In others the tubules are dilated and lined by hyaline glass-like cells, and in others the epithelium has undergone degeneration, granular or fatty. The renal vessels and their branches are much thickened, mainly owing to the great increase in thickness of the sub-endothelial layer due to an abundant formation of loose connective tissue. The elastic tissue is also seen to be increased, but whether this is dependent on the splitting of Henle's membrane or on the formation of fresh elastic tissue is doubtful. There is also a considerable amount of periarteritis, causing thickening of the external coat. The state of the middle coat varies; in some instances this also is fibroid, in others it is atrophied, and it is said in some cases to be hypertrophied. The thickening in the sub-endothelial layer of the inner coat is not uniform in its distribution, and may affect only a portion of the circumference of the vessel.

Cystic kidneys.—Cysts of the kidney are met with in one of three forms: (1) a single large cyst, usually involving one extremity

of the organ, in all probability a retention cyst, and produced gradually by obstruction involving some of the uriniferous tubules. Even when such a cyst is as large as an orange the remaining renal substance is often quite healthy. (2) Scattered cysts of varying size distributed throughout both organs, varying in size from a pin's head to a cherry, and usually more abundant in the cortex of the organ. These cysts, though occasionally present in the healthy kidney, are more frequently found in the granular kidney and in the contracted form of Bright's disease. In rare instances the formation of such cysts in granular kidneys is extreme, so that the organ has a general cystic appearance on section. In almost all cases of granular kidney microscopic cysts of a similar character may be found, which are probably retention cysts dependent on obstruction, and are merely dilatations of portions of the renal tubules. Their contents consist of a clear amber coloured fluid containing small quantities of urea. Blood and altered blood pigment may be present, causing the contents to be red or even black in colour. (3) General cystic disease of the kidneys. In this condition the kidney is apparently replaced by masses of cysts, but remains of the renal substance are to be found in the material separating the individual cysts.

Two conditions as regards causation are generally recognised: one where the affection is clearly congenital, and the second where, at any rate, the disease does not manifest itself until adult life, or middle age, or even later; the morbid anatomy of the two forms of the disease is, however, similar. The etiology of general cystic disease is not certainly known; it has been supposed to be due to obstruction of the renal tubules during fœtal life. Other observers have concluded that the condition is really dependent on anomalous development owing to the persistence of portions of the Wolffian body. Others, again, have looked upon the disease as of the nature of a new formation.

In congenital cystic disease malformations of the lower urinary tract, of the large intestine, and even of other parts of the body, such as the fingers and toes, are not uncommonly present. In the form of the disease seen in the adult cysts may be present in the mucous membrane of the pelvis of the kidney, of the ureter, and also in other organs. It is obvious that many of these phenomena cannot be explained on the hypothesis that cystic disease of the kidney is the sequel of atresia of the straight tubules, and the facts would seem to point to the conclusion that the malady is either of congenital origin, or that it is somewhat similar to a new growth.

In favour of the last view is the fact of the occurrence of some similar cysts in the liver and brain in some cases of cystic disease of the kidney. The usual bilateral character of the affection, however, is an argument that may be adduced against this view.

The wall of a single large cyst is in part formed by renal substance, and such cysts are usually situated more towards the anterior than towards the posterior surface of the kidney. The presence of such a cyst may be associated with ill-formed kidneys, but the rest of the kidney substance is generally of normal appearance. The fluid in the cyst is albuminous and contains only small quantities of urea and other nitrogenous extractives.

In general cystic disease both kidneys are usually affected, although in rare instances the disease has been unilateral, or more frequently has advanced to a considerably greater extent on one side than the other. The organs increase greatly in size and retain but little of their normal shape, consisting as they do of an enormous mass of cysts of varying size, from one-eighth of an inch in diameter to three-quarters or more. The cysts are thin walled and usually of a yellowish colour, owing to their contents consisting of a pale yellowish albuminous fluid containing traces of urea. Some of the cysts may be of darker colour, owing to the occurrence of hæmorrhage into their contents and the consequent decomposition of the extravasated blood. The cysts are entirely distinct from one another and cannot be emptied by pressure, as they do not communicate with the pelvis of the kidney.

The cyst wall consists of a delicate layer of connective tissue lined by flattened epithelium, and the remains of the kidney substance is found between the cysts, and thus there is frequently a great deal more renal substance even when both kidneys are affected than at first sight appears. In some instances the remains of the kidney substance present all the characteristic lesions seen in the granular kidney; in others it is fairly normal. Small cysts may be found underneath the mucous membrane of the pelvis of the kidney and even in the ureter, and somewhat similar cysts may be present in other organs, especially the liver and the brain.

Renal new growths.—The kidney is not often the seat of primary growths, and even in cases of malignant disease secondary deposits are not so common in this organ as in many others. Amongst benign growths adenomata are perhaps the most common. Small growths, from the size of a split pea to a pin's head, are not rare in otherwise healthy organs and in cases of granular kidney. In

some instances adenomata are associated with the presence of cysts, and rare instances have been described where extensive adenomatous deposits have undergone a cystic degeneration, producing in this way some resemblance to ordinary cystic disease of the kidney. Fibromata, lipomata, lymphadenoma, leuchæmic deposits, angiomata, and myxoma are occasionally found in the kidney. Adrenal rests are not infrequently found in the cortex of the kidney just beneath the capsule, and growths, usually malignant, but of varying degrees of malignancy and of an adenomatous structure, may derive their origin from such rests. Sarcomata, carcinomata, and epitheliomata all occur in the kidney, although the last is not so common as the former two. In rare instances epithelioma of the squamous variety has been found beginning in the kidney.

Sarcomata occur most frequently in the young, and in some instances are congenital, and the tumour may be of large size at birth or within a few weeks of birth. These congenital sarcomatous tumours have a very mixed structure, and portions of the tumour contain elements of a glandular type, so that the tumour is more accurately described as a malignant adenoma than a sarcoma. In some instances striped muscular fibre is also found in the tumour mass in some abundance, and to such tumours the name of rhabdomyoma has been given. Melanotic sarcoma is occasionally present in the form of secondary masses.

Carcinoma rarely occurs before the age of forty, and oftener in males. It is usually met with in one of two forms; in the one it is of the encephaloid variety, and in the other the arrangement of the cells is more gland-like, so that the growth might be described as a malignant adenoma. It is usually soft, and degeneration of large portions frequently occurs. It is rare for the overgrowth of fibrous tissue to be sufficient in amount for the tumour to be described as a scirrhus.

The presence of calculi has been thought by many to predispose to the formation of carcinoma, and certainly carcinoma and calculi are not infrequently associated, much as cholelithiasis and cancer of the gall-bladder.

In malignant disease the cortex is usually the first part affected, and even when the growth reaches a very large size the kidney maintains the general shape of the healthy organ. In the rare instances of epithelioma the disease begins in the pelvis and calices of the kidney, and spreads from these to the kidney substance generally.

Carcinomatous growths tend not only to involve the kidney

extensively, but may grow into the renal vein, and so cause thrombosis or lead to the dissemination of secondary growths.

The malignant growths beginning in adrenal rests are characterised among other things by the fact that they are frequently separated from the rest of the renal substance by a capsule, so that they can be shelled out even when of considerable size. Such growths often contain large quantities of fat, but when this is removed the fact can be made out that in general structure these adenomata resemble the normal suprarenal body.

Renal Tuberculosis.—Tuberculous disease may affect the kidney in association with other portions of the genito-urinary tract, and this is more especially the case in the male, or as part of a general acute tuberculosis, or the kidney may be the sole organ involved. Where the kidney is diseased together with other portions of the genito-urinary tract, the infection may be an ascending or a descending one; in the former case the primary mischief affects the bladder, testicle, epididymis, prostate, and the kidney either becomes involved as a result of a lymphatic infection or else owing to direct extension of the mischief up the ureter. In some instances of tuberculous disease of the bladder and prostate the ureter undergoes great thickening, and the mischief spreads to the kidney by direct extension; this, however, is rare. The co-existence of tuberculous lesions in the kidney with those in the lower urinary tract may sometimes, if not in the majority of cases, be described as the result of a descending infection, the tuberculous deposit in the kidney being really the primary source of the mischief. Modern writers hold the opinion that the infection is more usually descending than ascending, owing to the fact that in some 40 per cent of the cases of tuberculosis of the genito-urinary tract the kidney alone is affected, whereas the prostate or bladder is the only part affected in some 2 per cent. Where the disease is primarily renal in origin one kidney only may be affected, where it occurs secondarily to disease of the lower urinary tract both organs are often involved.

Scattered miliary tubercles may be found disseminated in the kidney in general tuberculosis. They are usually most abundant in the cortex, just below the capsule, but they may be found scattered throughout the organ. In the localised tuberculous lesions of the kidney pyelitis is frequently present, a number of scattered tubercles being found in the mucous membrane of the renal pelvis, and the lesion may spread from this point widely both into the kidney substance and down the ureter. The mucous membrane of the renal pelvis and of the ureter is swollen and thickened, and caseous masses

of considerable size may be found leading to the formation of tuberculous ulcers. The thickening of the mucous membrane in the upper part of the ureter may be of sufficient extent to cause hydro- or pyo-nephrosis, and the mucous membrane generally is intensely injected and villous in the areas between the ulcers. Masses of caseous tubercle of varying size may be found in the kidney substance itself, especially in the medullary portion in the vicinity of the renal pelvis. In many instances the destruction of the kidney substance in this way is so great that the organ is little more than a bag, the wall of which is formed by the thickened capsule and the remains of the cortical substance. The masses of tuberculous deposit are not restricted to the medulla, but may also be found in the cortex, and it is not uncommon for some of these to undergo calcification. The disease may be limited to one kidney, but in many instances it is bilateral, more especially when associated with tuberculous lesions of the lower urinary tract.

If tubercle bacilli are introduced into the circulation they not only may become arrested in the kidney, both in the glomeruli and in the interstitial tissue, but they may be excreted by the organ and found free in the tubules and even in the urine. Many infecting agents and toxic substances when introduced in the blood may in the course of their excretion produce a pyelitis, and it is possible that tuberculous pyelitis may sometimes arise in this way, and be due to the excretion of the virus of tubercle in the urine.

Tuberculosis of the kidney is more common in the male than in the female, according to some writers, in the proportion of three to one. It is most common in young adults, but it may occur at any age.

Pyelitis.—Inflammation of the pelvis of the kidney is usually of septic, tuberculous, or calculous origin, and in most cases the infecting agent reaches the pelvis of the kidney either by excretion through the kidney or else by ascending from the lower urinary tract. It is possible, however, that some cases of pyelitis arise as a result of the infecting agent reaching the organ through the blood-vessels of the pelvis of the kidney.

Both in tuberculous and in the various forms of septic pyelitis the disease may spread by direct and continuous extension from the urinary bladder. Any condition which leads to dilatation of the ureter will assist the ascent of infecting agents from below. The dilatation may be consequent on the impaction of calculi, or be the sequel of enlarged prostate, or else it may be of congenital origin. Slight degrees of pyelitis are not uncommon as complica-

tions in various acute specifics, *e.g.* typhoid fever. A number of substances such as turpentine, cantharides, not only can produce nephritis, but may also produce varying degrees of pyelitis. This is a point of some importance, as tending to show that the presence of certain substances in the blood may possibly produce pyelitis owing to their action on the mucous membrane of the renal pelvis. Acute pyelitis is certainly occasionally seen in the human subject where there is no reason to suppose that it is dependent on septic, tuberculous, or calculous causes, and it occurs, like other acute diseases, suddenly and during good health and without any evidence of implication of the kidney. Cases of primary pyelitis such as these are not very uncommon, and the infecting agent in such cases very possibly reaches the renal pelvis through the blood stream. It has been supposed that pyelitis may result from exposure to cold, but there is no very definite evidence in support of this view. It is often associated with the presence of calculi, or of gravel, and more especially with those consisting of uric acid.

Pyelitis where it complicates cystitis is not uncommonly of gonorrhœal origin, and such cases may run a very acute course, and are liable to lead to pyelo-nephritis, abscess of the kidney, and perinephritic abscess.

The etiology of the cases of primary abscess of the kidney is very obscure when not dependent upon morbid processes in other organs, *e.g.* the heart, leading to embolism. It has been supposed to follow injury and also exposure to cold, but these are clearly only predisposing causes, the actual exciting cause being a microbic invasion of some kind dependent on the presence of organisms in the blood.

Suppuration in the loose cellular tissue surrounding the kidney may arise primarily as the sole morbid condition, or it may be a complication of, and associated with suppuration in the kidney itself. In the latter cases it is especially associated with abscess of the kidney and pyelitis.

Cases of acute perinephritic abscess are sometimes seen arising quite suddenly in previously healthy persons, and these cases present a considerable resemblance to the somewhat analogous instances where an acute pyonephrosis or acute pyelitis arises under similar conditions and without the presence of any such lesions as calculi or tuberculous deposits. All these cases are probably dependent upon blood infection, the exact nature of which is not definitely known, but is presumably microbic.

The morbid anatomy of pyelitis presents considerable differences

dependent on the presence or absence of such complications as pyelo-nephritis or pyonephrosis.

In uncomplicated pyelitis the mucous membrane of the renal pelvis is swollen and submucous hæmorrhages are scattered here and there. The portion of the mucous membrane superficial to these hæmorrhages may slough, and thus ulcers of varying size are formed. The surface may be covered with a layer of pus, and in some instances a membranous exudation may be present. The pelvis and calices frequently undergo dilatation, and pyonephrosis is not an uncommon complication. In such cases the kidney becomes distended owing to the flattening of the pyramids, and the capsule of the organ becomes considerably thickened and often adherent to the surrounding structures. Perforation of the wall of the renal pelvis or of some part of the sac of the pyonephrosis may take place, leading to the complication of a perinephritic abscess. In pyelo-nephritis foci of suppuration are found in the medullary portion of the kidney extending into the cortex, and small abscesses are frequent just beneath the cortex. In pyonephrosis and pyelo-nephritis necrosis of portions of the pyramids may occur, and the renal pelvis may contain fragments of disintegrated and necrotic renal tissue.

Calculi, even of large size, are not uncommonly present in the renal pelvis, not only without producing any symptoms, but also without leading to any marked anatomical changes in the organ. Pyelitis is, however, the most usual complication, and when this is dependent on the presence of calculi, the mucous membrane of the renal pelvis is often villous and greatly thickened, and the mucous membrane may be ulcerated. In some cases the calculi are not only of large size, but branch so as to fit the calices, or numerous facetted stones may be present.

Renal calculi may lead to the production of hydronephrosis, pyelo-nephrosis, pyelo-nephritis, or atrophy. In atrophy of the kidney associated with the presence of stones the atrophy may not be real, but the kidney may remain as a shrivelled sac, the remains of a condition of former hydronephrosis. In other instances the atrophy is more perfect, the general form of the kidney being maintained, but the organ is greatly reduced in size and frequently fibroid. Hydronephrosis can be produced experimentally by obstruction of the ureter, and subsequent drainage of the distended kidney may be followed by the production of a very perfect atrophy.

The impaction of a calculus in the renal pelvis or ureter is not always followed by the production of hydronephrosis. It may lead

to the complete suppression of urine. Renal calculi are sometimes associated with malignant disease of the kidney.

Nephrolithiasis.—The formation of calculi in the calices of the kidney and in the renal pelvis may be dependent on a disturbance of the general metabolic changes of the body, or else it may result as a sequel to morbid processes limited to the renal pelvis.

(1) Disturbances of metabolism may affect the amount of uric acid, oxalic acid, or other substance present in the urine, or else, owing to alterations in the reaction of the urine, the form in which the salts of these acids are excreted may be altered. In this way the deposition of anomalous crystals may take place and so form the nucleus of a calculus. Cystin and indigo are substances which in rare instances have formed the bulk of a renal calculus, and they may be present in the urine in largely increased amount owing to metabolic disturbances.

Uric acid is the most common constituent of renal calculi, or at any rate in most instances it forms the nucleus of such stones. When the formation of a uric acid calculus is dependent on a disturbance of metabolism there are at least two modes of origin of such disturbance, inasmuch as uric acid has a double source. A portion of the uric acid excreted is formed directly from the metabolism of the tissues themselves, another portion from the metabolism of certain proteid constituents of the food. Under a suitable diet, such as milk, some vegetables, eggs, and cheese, the excretion of uric acid is much diminished, and practically the whole quantity present in the urine is then derived from the metabolism of the tissues. With a large meat diet, on the other hand, the excretion of uric acid may be greatly increased owing to the presence in the food of the proteid substances from which uric acid is derived. It is most important to recognise that with a suitable diet the formation of uric acid in the body can be greatly diminished, and in fact restricted to that which owes its origin to the metabolism of the tissues. It is clear from this that an excess of uric acid in the urine may arise theoretically either as a result of primary disturbance of the metabolism of the tissues, in which case it cannot be influenced to any great extent by diet, or, in the second place, owing to an ingestion of diet rich in alloxur bases. The formation of calculi, therefore, containing uric acid may be dependent on at least two disturbances of metabolism, quite apart from the fact that it may be independent of these, and due solely to alterations in the urine, such as variations in the acidity.

Oxalate of lime is not such a common constituent of urinary

calculi as uric acid, although it is by no means rare, and it is remarkable owing to the fact that it leads to the formation of peculiarly dense calculi. Metabolism of the oxalates is totally different to that of the urates, inasmuch as there is no evidence to show that oxalates are a product of the normal metabolism of the tissues of the body, in other words, all the oxalates of the urine are derived from the presence of oxalic acid in the form of oxalates in the food, and especially in certain vegetables, *e.g.* rhubarb, tomatoes, etc. Thus, although a uric acid diathesis may exist, it would not be equally correct to assert the existence of an oxalic acid diathesis. Although oxalates are not formed in the healthy organism as a result of the metabolism of nitrogenous or carbohydrate tissues, there is no doubt that oxalates may be formed as a result of decomposition and fermentation of the contents of the stomach and intestine. The oxalates of the urine would bear somewhat the same relationship to the processes of gastric digestion as the sulphates, and especially the aromatic sulphates, to intestinal digestion. The formation of oxalates in abundance and their subsequent excretion are more associated with derangement of the gastric processes than with intestinal disorders. The formation of organic sulphates, on the other hand, is more especially associated with intestinal decomposition. In some instances the formation of oxalates takes place in great abundance, as the urine may deposit large quantities of oxalate of lime easily visible to the naked eye. In most cases, however, the quantities are much smaller and the crystals are only detected upon microscopic examination.

The deposition of *phosphates* in the urine is also occasionally associated with metabolic disorders, although there is some doubt whether a condition of true phosphaturia exists. Most cases of so-called phosphaturia are instances where the relative proportion between the soluble and the insoluble phosphates is altered in the direction of increasing the amount of earthy phosphates. Certain persons, however, invariably pass a urine so faintly acid or even alkaline that it always contains phosphates suspended, and such persons are liable to the formation of primary phosphatic calculi. In rare instances this is associated with the formation of *calcium carbonate* calculi. The metabolic disorder giving rise to this condition is not one necessarily affecting the phosphatic metabolism of the body, it may be entirely dependent on an anomaly in the reaction of the urine. Inasmuch as this is dependent on the presence of acid phosphate of soda, derived in great part from the fleshy food, it is indirectly connected with phosphate metabolism.

(2) Although the formation of calculi may be associated with disturbances of metabolism leading to the presence of increased amounts of uric acid, oxalic acid, or insoluble phosphates in the urine, this is by no means the sole mode of origin of urinary calculi. Their formation may depend not so much on the actual amount of the constituent forming them present in the urine, as on the mode of its crystallisation, or on the absence of some other constituent in the urine which normally keeps the particular constituent in question in solution.

Uric acid is present in the urine in the form of a urate, either as a bi-urate of soda or a quadri-urate. Its existence in this form is intimately associated with the amount and reaction of the phosphates present, as a mixture of bi-urate of soda and acid phosphate of soda tends to undergo decomposition with the liberation of free uric acid and the formation of the neutral phosphate. A urine poor in saline ingredients tends to deposit free uric acid notwithstanding the fact that the total amount of uric acid present may not be large. On the other hand, a urine loaded with uric acid may contain it all in the formation of a soluble urate, provided the urine contain abundant saline constituents. It is notorious that dilute urines frequently tend to deposit free uric acid, and that concentrated febrile urines deposit urates. No more striking instances could be quoted showing how the quantity of saline constituents and pigments modifies the precipitation of uric acid quite apart from the mere amount of this substance present.

Uric acid calculi are frequently associated with errors of diet, more especially the consumption of large quantities of meat. Under such circumstances the deposition of uric acid may occur owing to the presence of an increased amount of it in the urine, or else as a result of the increased acidity of the fluid. Uric acid calculi, however, are by no means confined to large meat eaters, but occur with considerable frequency in the poor. It is supposed that under these circumstances their origin is associated with the lack of saline constituents of the urine, owing to the diet being poor in salts, and this is also asserted to be the cause of the frequency of calculi in certain parts of India and China.

The oxalate of lime calculus is associated, as far as is known, with gastric disturbance, indigestion, dilatation of the stomach, and the consequent formation of oxalic acid in considerable quantities owing to the decomposition of the gastric contents. The urine normally contains a sufficient quantity of calcium to combine with all the oxalic acid present, and thus precipitate it in the form of

oxalate of lime; the acid phosphate of soda normally present in the urine prevents this formation of oxalate of lime, and thus the deposition of calcium oxalate, like that of uric acid, may be associated with the variations in the amount of the acid phosphate present, although the relationship is an opposite one in the case of uric acid to what it is in the case of the oxalates, an excess of acid phosphate tending to precipitate the former and to keep the latter in solution.

The form of crystallisation of uric acid, and to a greater extent that of oxalate of lime, is profoundly influenced by the presence of colloid substance in the urine, such as blood, mucus, pus, albumin, etc. The presence of a small quantity of such colloid material in a solution from which oxalate of lime is crystallising out may lead to the formation of very large crystals of this substance. This may have an important bearing on the mode of formation of the nuclei of calculi, and it has been shown that, at any rate in the formation of oxalate of lime calculi, the nucleus may consist of a single large crystal. In other instances the nuclei of calculi may consist of organic matter, such as blood, etc. It is possible in this manner to explain the formation of calculi in the pelvis of the kidney, as it is conceivable that a slight pyelitis, produced perhaps from other causes may, owing to the exudation of blood or proteid matter, lead to the formation of abnormally large crystals of the urinary constituent, and so lay the foundation of the calculus. In some instances the pyelitis is doubtless produced by the irritation caused by the ordinary crystallisation of a particular urinary constituent, such as uric acid or oxalate of lime. There is no difficulty in understanding the subsequent growth of a calculus once its nucleus has been formed.

The main etiological factors, therefore, in the production of calculi are, firstly, disturbances in the metabolic processes of the body; and, secondly, alterations in the urine not associated necessarily with any variation in the amount of the particular ingredient excreted, but influencing the crystallisation and deposition of the particular substance. It is a matter of considerable importance in the treatment of urinary calculi that their formation is not entirely dependent on a mere increased amount of the particular constituent involved.

Calculous disease is more common in men than in women, and may occur at almost any period of life, but is especially frequent in youth. In childhood uric acid and urates are the most common constituents of calculi. Oxalate of lime does not occur so

frequently in the young as in the adult and middle aged. It is not uncommon for calculi to be mixed, and even to consist of alternate layers of uric acid and oxalates.

Hydronephrosis is a condition in which the calices and pelvis of the kidney are distended to varying extents owing to obstruction to the exit of urine along the ureter or lower urinary tract. In some cases hydronephrosis is *congenital*, and then is usually bilateral, and associated with some malformation of the lower urinary tract, and in some instances with such a condition as extreme phimosis. In other cases the obstruction may be situated where the ureter opens into the bladder, the latter viscus being normal. *Acquired* hydronephrosis may depend on a variety of causes, one of the most frequent is the impaction of a calculus in the ureter; such impaction not uncommonly takes place in the portion of the ureter passing through the wall of the bladder. In other instances the stone becomes blocked in the upper third of the ureter. But the impaction of a calculus in the ureter is not invariably followed by the production of hydronephrosis, as complete suppression of urine may ensue. The ureter may become blocked not only by calculi, but also by the detachment of portions of growth or of masses of tuberculous and caseous renal tissue. In some cases it would appear as if the mere inflammatory swelling of the mucous membrane of the ureter were sufficient to cause complete obstruction to the exit of urine, but under these circumstances a pyonephrosis is more often produced than a hydronephrosis.

Hydronephrosis often results from anomalies in the kidney and its vessels. Thus it is a fairly common complication of movable kidney, the displaced organ in some patients being associated with kinking of the ureter as it emerges from the renal pelvis, and this may be sufficient to produce a hydronephrosis. In other instances there is some anomaly in the mode of attachment and form of the ureter: thus it may be exceptionally narrow or set at an acute angle with the renal pelvis, or the renal pelvis may be congenitally dilated and somewhat pyriform, so that the urine does not find a ready means of exit from the dilated pelvis into the ureter. The renal arteries may also be anomalous, and an accessory one may pass over the ureter in such a manner as to kink it, and so cause obstruction.

In all these conditions the hydronephrosis is usually unilateral, but bilateral hydronephrosis may be produced by the pressure of tumours, generally pelvic in position, on the ureters. This is especially the case in uterine carcinoma, and is occasionally seen in

fibroids. The adhesions formed as a result of peritonitis, especially pelvic peritonitis, may also lead to the obstruction of the passage of urine along the ureter.

The hydronephrosis produced by any of these mechanical causes may be transitory, persistent, or intermittent. Movable kidney is especially a cause of intermittent hydronephrosis. In very exceptional instances, where the hydronephrosis has been dependent on pelvic peritonitis, and perhaps on uterine fibroids, the hydronephrosis has not only been intermittent, but has occurred synchronously with the occurrence of the menstrual periods. It is supposed that in such cases the swelling of the pelvic viscera during the catamenia has led to a slight increase in the obstruction, produced may be by peritoneal adhesions, and has thus caused temporary complete obstruction.

In hydronephrosis the renal pelvis first undergoes dilatation, and this is soon followed by the flattening of the apical portions of the pyramids. Subsequently the pyramids themselves become flattened and compressed, and the whole kidney becomes distended, the kidney tissue becoming spread out to form the sac of the hydronephrosis. In extreme instances the kidney may be distended to such an extent that this sac may not amount to more than one-eighth of an inch in thickness. The connective tissue forming the walls of the calices and accompanying the vessels as they pass up between the pyramids remains and forms partitions between the loculi of the hydronephrosis. Even in the most extreme cases the destruction of renal tissue is more apparent than real, as a large amount of it remains spread out, forming the sac. The glomerular chambers in the earlier stages become considerably dilated, but in old-standing cases, owing to the overgrowth of connective tissue beginning in the interstitial tissue of the kidney, the glomeruli may become fibroid, thickened, and atrophied. The epithelium lining the tubules undergoes considerable changes, in many places being shed, and in others undergoing degeneration and becoming hyaline and glass-like. Large numbers of tubules undergo atrophy and disappear.

The fluid in a hydronephrosis, especially if of long duration, contains but a low percentage of urea and other nitrogenous extractives, and there may be in addition traces of albumin. In some instances hæmorrhage may occur into a hydronephrotic sac, especially when this is dependent on the presence of calculi. If the hæmorrhage be copious a solid tumour consisting of the sac-like kidney distended with blood clot may be produced, and the clot may undergo secondary changes such as calcification.

General Symptomatology of Renal Disease

Morbid processes affecting the kidney not only produce changes in those organs, but in addition they cause in many instances widespread effects on other organs and tissues of the body. Some of these general effects are produced by all diseases which seriously involve the kidney substance, others are only seen in certain affections of the kidneys. Further, there is an intimate relationship between renal diseases and the integrity of the nervous system, as nearly all forms of grave renal disease are liable to be complicated by the development of toxic symptoms which are known as uræmia. Many of the so-called general symptoms of renal disease, such as headache, vomiting, are really of toxic origin, and are therefore in a certain sense uræmic.

The effects produced may be classified as follows: (1) changes in the composition of the urine; (2) cardio-vascular changes; (3) dropsy; (4) uræmia (see Vol. II. p. 81); (5) disorders of nutrition; (6) diminished resistance of the tissues, leading to the liability to so-called septic complications.

The changes produced in the urine as a result of renal disease necessarily take one of two forms—either the amount and proportion of the normal constituents are altered, or abnormal substances, such as blood, albumin, casts, etc. are present in the urine as a result of the anatomical changes produced in the organ by the morbid process. The alteration in the amount of the normal constituents, however, may be dependent on more complex causes. In many renal diseases the flow of urinary water may be increased in amount; this has commonly been looked upon as dependent on the increased arterial tension produced by the accompanying cardio vascular changes, but it may be dependent simply, as suggested above, on the mere diminution in amount of the kidney substance. In other instances the increase in the flow of water may be dependent on the subsidence of dropsy produced by the renal disease. Not uncommonly it is in part dependent on the patient being put on a milk diet, and is merely the result of the diuretic action of this substance. Thus the increased flow of water may be due to the anatomical changes in the kidney itself, or else to functional or structural changes in other parts of the body.

Although the presence of abnormal substances in the urine is usually dependent on the anatomical changes produced by the disease in the kidneys, it is possible that proteids may be present in

the urine without grave structural lesions. This is seen, for instance, in the cases of so-called physiological albuminuria. Albumoses may also occur in such instances, although when present in abundance in renal disease serious organic disease elsewhere is usually to be found.

Urinary water.—Diseases in which the circulation through the kidney is hindered either by active or passive congestion are characterised by the secretion of a scanty urine, inasmuch as in both these conditions the rate of flow of blood through the organ is diminished. Further, in the diseases accompanied by dropsy the urinary flow is also necessarily diminished, at any rate during the onset and height of the anasarca. In maladies accompanied by destruction of the renal substance the urinary flow may be either diminished or increased in amount, and even in cases where there is but little renal substance left the excretion of urinary water may be abundant, *e.g.* granular kidney, the contracted form of Bright's disease, cystic kidneys, and some forms of lardaceous disease. In chronic Bright's disease, and especially in the large white kidney, the flow of urine is usually diminished in amount, but this is commonly dependent on the co-existence of dropsy, but where dropsy is absent the flow may not be greatly below the normal, and is not uncommonly above it.

In all renal diseases accompanied by an increased excretion of urine the specific gravity of the urine is considerably below the normal. All observations on the specific gravity, however, should be made on a mixed sample of the twenty-four hours' urine.

Suppression of urine may occur in a considerable number of renal affections, and it is customary to recognise two varieties, the so-called obstructive and non-obstructive. In the former a gross lesion, such as the presence of a calculus, is found blocking the urinary tract, but non-obstructive suppression is usually accompanied by the presence of serious disease of the renal substance, such as acute nephritis, or of chronic destructive lesions, as in the later stages of chronic Bright's disease, cystic kidney, or tuberculous kidney. In some instances complete suppression may take place as the result, apparently, of reflex influences produced by disease in other parts of the body, and the kidneys may present no signs of gross disease. The clinical picture produced by obstructive suppression differs from that seen in the suppression occurring as a terminal symptom in chronic renal disease. The latter produces ordinary acute or chronic uræmia, and the former that variety of uræmia which has been described as latent. Latent uræmia, indistinguishable from

that seen in calculous obstruction, may be seen as an accompaniment of complete suppression, either of reflex origin or produced by complete arrest of the renal circulation, as in thrombosis of the renal arteries. The suppression of urine accompanying such maladies as diphtheria, and produced by the action of toxic substances, may also sometimes resemble in its clinical symptoms obstructive suppression. It is a matter of some clinical importance that the symptoms characteristic of obstructive suppression may occur in conditions in which the kidneys themselves are not seriously affected.

Urea.—The excretion of urea is often diminished in renal disease, although this diminution is not always dependent on an incapacity on the part of the kidney to excrete, but may be associated with the diminished metabolism dependent on impaired appetite and the presence of diarrhœa and vomiting, as these are frequent accompaniments of many forms of renal disease. Considerable quantities of albumin are also often present in the urine in renal disease, and this will necessarily tend to diminish proteid metabolism. All these causes will operate to diminish the amount of urea excreted, and observations, either on the percentage of urea or total amount excreted, are of comparatively little value unless controlled by observations on the ingesta and on the proteid waste.

In many forms of chronic renal disease there is considerable impairment in the excretion of urea owing to great destruction of the convoluted tubules, and in such cases the amount present in the blood and tissues may increase twenty-fold.

A sudden diminution in the quantity excreted is often looked upon as one of the warning signs of the imminence of uræmia.

The excretion of *urinary pigment* is usually deficient in renal disease, especially in the chronic varieties, so that the urines are pale.

In chronic disease there may be some delay in the excretion of such substances as iodide of potassium and methylene blue in the urine after their administration, and in this way a rough estimate as to the efficiency of the kidney as a filter may sometimes be obtained.

Abnormal substances.—The most important abnormal substances present in the urine in renal diseases are blood, pus, proteids (albuminuria), and casts.

Blood may be present either in mere traces or in sufficient quantity to cause the urine in the renal pelvis, the ureter, and the urinary bladder to undergo clotting. When the blood is derived from the kidney blood casts are usually present in the urine, but

copious hæmorrhage may take place from the mucous membrane of the renal pelvis, ureter, or lower urinary tract, and in such cases blood casts are necessarily absent. Hæmorrhage from the kidney substance itself is usually dependent on either active or passive congestion, and, therefore, is especially seen in infarction, acute nephritis, and the passive congestion of heart disease.

Usually the quantity of blood lost is small, but in some forms of acute nephritis and acute Bright's disease relatively large quantities, causing the urine to be uniformly red, are present. Copious hæmorrhage is sometimes seen in chronic renal disease, more especially in the granular kidney. In such instances it may be dependent on the occurrence of a complication, such as acute nephritis, but it is probable that the very free hæmorrhage sometimes seen is derived from the renal pelvis rather than from the kidney substance. In exceptional instances free hæmaturia is seen in cases of cystic kidney. Hæmaturia is usually a conspicuous symptom in malignant disease of the kidney, and also in pyelitis, whether of tuberculous or of calculous origin.

Where the quantity of blood is small, as in some forms of acute nephritis, the urine often has a smoky tint on account of the changes due to the decomposition of the blood pigment.

Pyuria.—The various forms of pyelitis on the one hand and tuberculous kidney on the other are the most common causes for the presence of pus in the urine, provided that the source of the pus is not the lower urinary tract. Occasionally it may owe its origin to the formation of renal abscess dependent on embolism. Pyuria is sometimes due to the rupture of an abscess into some portion of the urinary passages. When the pus is of renal origin, as in renal abscess, casts consisting of pus cells may be found.

The diagnosis of the source of the pus has to be made partly on the symptoms, partly on the characters of the urine, and more especially its reaction and other accompanying phenomena, as, for instance, the presence of prostatic threads. Valuable information may sometimes be obtained by determining whether the pus is uniformly mixed with the urine, or whether it comes at the beginning or end of micturition, the latter being common where the pus is derived from the lower urinary tract, such as the urethra and prostate. In pyelitis the urine is usually acid, and in cystitis alkaline, but neither of these statements is true of all cases. Where pus is present in quantity the urine will necessarily contain a certain amount of albumin derived from the liquor puris. But such diseases as calculous and other varieties of pyelitis may be

accompanied by secondary waxy degeneration, and this will cause a further albuminuria. In examining such urines it is advisable to let the pus subside, or to separate it by centrifugalising before testing the supernatant liquid for albumin. In cases of uncomplicated pyelitis the urine must contain some albumin, but the quantity will not be very large unless the pus be extraordinarily abundant, or a renal complication, such as lardaceous kidney, be present.

Albuminuria.—Small quantities of albumin are found in the urine of apparently healthy persons in a considerable proportion of cases, if the urine be tested with ordinary reagents such as nitric acid. Very minute traces of proteids may be present in as many as 30 per cent of apparently healthy persons. This so-called physiological albuminuria is more especially marked in male adolescents, and it may either be intermittent or constant in its appearance. If intermittent it is especially liable to be marked on first rising in the morning, the so-called postural variety, or after meals, or after severe exercise, or after cold bathing. Even in cases where it is constant in its presence it is increased in amount by any of these actions. In most cases of physiological or functional albuminuria the presence of the albumin is not constant. Casts, or at any rate casts containing definite renal elements, are absent, although on centrifugalising the urine a few hyaline cylinders may be found. These patients present no other symptoms or signs of renal disease, and the condition not uncommonly completely disappears when they reach adult life, although this is not invariable. It has been thought by some that patients suffering from physiological albuminuria are more liable to become affected by such diseases as Bright's disease. This, however, is not certain, though for some reasons probable.

Most renal diseases are accompanied by albuminuria, although the amount of proteid matter present in the urine varies greatly. Thus in the true granular kidney a mere trace may be present, and, on the other hand, in the lardaceous kidney and in the contracted form of Bright's disease large quantities of urine containing a high percentage of albumin may be passed, so that as much as forty grammes of dry proteid may be lost in the patient's urine per diem. It is probable that in the granular kidney the albuminuria is not absolutely constant, and that during certain periods the urine may be free from albumin, notwithstanding the existence of this serious renal disease. In very rare instances albuminuria may be absent even in fatal cases of the contracted white kidney, and the writer

has seen three cases of fatal uræmia where this was the case, and where very extensive chronic renal disease existed.

The proteids present in the urine in renal disease are serum albumin and serum globulin, and it is not uncommon for traces of albumose to be present. Albumose in large quantities has been described in some cases of granular kidney. In a small number of these extensive disease of the marrow of bones, so-called myeloma, has been present, but albumosuria is seen in granular kidney without the presence of this rare disease. Albumosuria also occurs in empyema and acute pneumonia.

The greatest percentage loss of albumin occurs in cases of acute Bright's disease and in the large white kidney, but the actual amount is often not very great owing to the great diminution in the quantity of urine excreted. In the contracted form of Bright's disease and in some varieties of waxy kidney the loss may be much greater, notwithstanding the fact that the percentage amount present may be less than in the diseases mentioned above. This arises from the fact that the quantity of the urine excreted in the lardaceous kidney and in the contracted form of Bright's disease is very much above the normal. The albuminuria of renal disease may undergo postural and dietetic variations similar in character to those seen in so-called physiological albuminuria.

In the active congestion accompanying acute specifics the amount of albumin present is usually small, and even in cases of infarction the quantity is not generally large. It is very characteristic of infarction for the albumin to appear suddenly and to disappear with equal suddenness. In passive congestion the amount of albumin is usually small, the only exception to this is in the albuminuria accompanying pregnancy, where the urine may become solid on boiling, but it is probable that this albuminuria is of toxic origin rather than dependent on simple passive congestion. It is unusual for the albumin to exceed one-third in cases of simple passive congestion. The distinction between the albuminuria of passive congestion and that due to nephritis must be made on other grounds than the mere amount of albumin.

Casts.—The term "casts" is applied to peculiar cylindrical bodies in the urine and in the renal tubules. They are in some instances apparently moulds of the interior of the renal tubules of variable size and length, and then present a hyaline or slightly granular structure. The material forming such casts or cylinders is proteid in nature, although not necessarily always fibrin, as was once thought to be the case. These hyaline casts are very

probably formed by the coagulation of proteid material extruded from the renal cells or passed out from the glomeruli, but it would seem that the protoplasm of the renal cells may under certain circumstances—*e.g.* the action of certain renal poisons—undergo a kind of solution of continuity and be extruded in drops, the coalescence of which forms the cast.

Casts, however, are not always hyaline, they may consist of agglomerations of definite cell elements; thus both blood and pus casts occur where the corpuscles are kept together by fibrin, or else the cells—*e.g.* pus cells—are embedded in and held together by the material forming hyaline casts. In some conditions casts consist of the renal cells—epithelial casts—showing the characteristic rodded cells of the convoluted tubules of the kidney, and here also the material binding the cells together may be the same as that forming hyaline casts. Sometimes epithelial casts are merely renal cells in greater or less abundance that have become detached from the basement membrane, and shed into the lumen of the tubule.

Casts may undergo degeneration or consist of elements that have undergone degeneration, and waxy and fatty casts afford illustrations of this. The latter are of very considerable clinical importance, and consist either of renal cells that have undergone fatty degeneration, or else they resemble hyaline casts, but contain droplets of fat in greater or less abundance. Casts hyaline in character, but yielding the lardaceous reaction, are occasionally found in the urine of the waxy kidney. Casts vary much in size, both as regards breadth and length, the former affording some evidence of the part of the kidney in which they have been formed. They are sometimes present in sufficient abundance in the urine to form a visible deposit, although this is exceptional, and more usually they have to be searched for in the deposit left by allowing urine to stand, or else by centrifugalising the urine. Albuminuria is usually associated with the presence of these bodies, and the nature of these may throw light on the cause of the albuminuria. Hyaline and granular casts are often found in the urine of the granular kidney, epithelial casts in acute and chronic Bright's disease, fatty casts in chronic Bright's disease, and pus casts in pyelo-nephritis and abscess of the kidney. Blood casts are found in both active and passive congestion of the kidney, and their presence is often the best evidence that the hæmaturia is of renal origin.

The mere presence of casts in the urine is not to be regarded

as unequivocal proof of the existence of serious organic disease, but fatty, blood, waxy, and pus casts always signify the presence of organic disease, and fatty casts may be evidence of very serious disease. On the other hand, hyaline casts are frequently present in small numbers in cases of so-called functional, physiological, or cyclical albuminuria, and also in cases of jaundice.

Cardio-vascular changes.—In many renal diseases secondary changes are produced in the cardio-vascular apparatus, especially in the small arteries and in the heart. These effects are seen especially in Bright's disease and in the granular kidney. They may occasionally accompany cystic disease of the kidney. The typical effects produced are on the one hand cardiac hypertrophy, and on the other thickening of the inner coat of the arterioles, owing to the great increase in the connective tissue, and possibly of elastic tissue, in the sub-endothelial layers of the inner coat. The lesions in the small arterioles vary a good deal, not only in their amount, but also in their distribution. They are, however, always well marked in the renal vessels, and in some conditions are equally well marked in all the vessels of the body. In disease primarily renal in origin these changes are best marked in the renal vessels. On the other hand, in some varieties of the granular kidney where the renal lesion is merely an accompaniment of the general arterial degeneration, not only may the vascular lesions be extremely widespread in their distribution, but the renal lesions may be, comparatively speaking, slight in amount.

The effects on the heart and on the arteries seen in Bright's disease are produced very rapidly—notable cardiac hypertrophy may be developed in as short a time as a month to six weeks from the onset of the Bright's disease. Synchronously with the development of the cardiac hypertrophy the pulse acquires the well-known high-tension characters which form so marked a feature of chronic Bright's disease. The pulse develops the characteristics of high tension before well-marked changes occur in the vessel wall capable of detection by the finger, so that it is not uncommon in renal disease for the radial artery to present the characters of the high-tension pulse, but yet for the vessel to be soft and its walls not appreciably thickened. In general arterial degeneration, and in the so-called arterio-capillary sclerosis seen with some forms of granular kidney, such a vessel as the radial usually presents obvious signs of thickening and fibrosis at a time when the patient first comes under observation.

The cardiac hypertrophy and arterial lesions were at one time

correlated with the destruction of renal substance, and were looked upon as directly dependent upon this. The association of these vascular lesions with the granular kidney was explained in this fashion. Mere destruction of the renal substance, however, does not lead to any special cardiac hypertrophy or increase in arterial tension. This is seen from the fact that cardiac hypertrophy and arterial degeneration may be absent in cases of cystic kidney, and in those anomalous cases of congenital malformation of the kidney which are associated with variable degrees of hydronephrosis and atrophy of the kidney substance. In both of these conditions the amount of kidney substance present may be very small, yet cardiac hypertrophy and a high tension pulse may both be absent. Instances are also occasionally seen where the cardio-vascular lesions are absent, notwithstanding the existence of granular kidney; this is more especially seen in cases occurring in young persons. Lardaceous kidney also is not accompanied by these cardio-vascular changes.

Cohnheim considered that the cardio-vascular lesions developed directly as a result of the destruction of the renal substance, and that the hypertrophy of the heart and the increased arterial tension were necessary in order to maintain the circulation with sufficient efficiency through the remaining kidney substance. There are, as just mentioned, difficulties in the way of acceptance of this view, and it is possible to regard the high tension as of toxic origin, though the nature of the toxic substance is unknown, and the vascular changes may be the sequel of a long continued high tension. In other words, the thickening of the arteries may not be the cause of the increased blood pressure, but may be the result. It has even been supposed by some writers that the initial phenomenon is the development of the cardiac hypertrophy in response to the presence of some irritant in the blood. It is difficult, if not impossible, to say at the present time whether the cardiac hypertrophy is primary and the arterial lesions secondary, or whether they are both phenomena dependent on the production and maintenance of an increased arterial pressure.

The cardio-vascular lesions produced are responsible for many of the symptoms seen in renal disease and for some of the most serious complications. The cardiac hypertrophy, especially, perhaps, in cases of granular kidney, is liable sooner or later to become complicated by the occurrence of cardiac dilatation, and the patient will then present the symptoms and physical signs of mitral regurgitation. In some cases where dropsy is not a

prominent symptom cardiac pain and palpitation are marked effects, and in such patients it is not uncommon for a rough rumbling presystolic murmur to be audible, or for the first sound to be reduplicated or muffled in character. Where dropsy becomes marked the case presents considerable resemblance to one of mitral disease, but the renal origin of the malady can often be detected by a careful examination of the pulse, the urine, and the fundus oculi.

Cerebral hæmorrhage is a frequent and very serious complication of the vascular lesions produced by renal disease, and in many of these cases the hæmorrhage is very copious owing to the existence of the high tension. This accident is perhaps on the whole more likely to occur in the cases where the general arterial lesions are highly developed than in those where the kidney lesions preponderate. Thus, in the two varieties of granular kidney cerebral hæmorrhage is more apt to take place in the form accompanying general arterial sclerosis than in that which is primarily renal in origin, and on post-mortem examination, in cases of cerebral hæmorrhage, the kidneys are more often large and granular than small, granular, and contracted. Still cerebral hæmorrhage may occur not only in both forms of granular kidney, but also in both varieties of chronic Bright's disease. It is important to recognise that cardiac hypertrophy and arterial lesions are not only well marked in the contracted form of Bright's disease, but also in many cases of the so-called large white kidney.

The degree of the development of the arterial lesions in cases of renal disease can often be gauged by an examination of the arteries of the fundus oculi, the thickening of the walls causing a double outline of the vessel. Further, the vessels, instead of running in a series of easy curves, are often acutely bent in their course, and it is for this reason that the term "wiry arteries" has been applied to the vessels of the retina in renal disease. In rare instances aneurysms may be seen, and very commonly flame-shaped hæmorrhages and patches of exudation are present (see Vol. III. p. 395).

The cardiac hypertrophy of renal disease is generally moderate in its amount, and, although in the earlier stages it involves mainly the left side of the heart, there is usually some enlargement of the right side. In uncomplicated renal disease the heart never reaches the size and weight that it does in valvular disease (see also Vol. IV. pp. 379, 419).

Anasarca.—Dropsy is a marked feature of many forms of renal

disease, and more especially of Bright's disease, and it is especially characteristic of acute Bright's disease and of that form of chronic Bright's disease spoken of as large white kidney. Dropsy is often absent throughout the course of that form of chronic Bright's disease described above as the contracted white kidney, and when it occurs in cases of granular kidney it is usually of secondary origin and dependent on cardiac failure, so that, though occurring in renal disease, it is cardiac rather than renal dropsy. Dropsy is often present in passive congestion of the kidney, and when it is seen accompanying this condition it is usually of cardiac origin, and dependent on general venous congestion. Dropsy is absent in cases of transitory nephritis complicating specific fevers. In waxy disease dropsy is often a prominent symptom, but in many of these cases Bright's disease is also present.

The occurrence and increase in amount of renal dropsy are invariably accompanied by a diminution in the excretion of urine, but this partial or even complete suppression of urine is not the cause of the dropsy, as is seen from the fact that the suppression of urine occurring in other renal disease, *e.g.* calculous obstruction, is not accompanied by dropsy, and this is usually absent even when complete suppression lasts for as long as a week or ten days. In exceptional instances of this long-continued suppression in calculous disease slight œdema may occur, but nothing comparable to the general dropsical swelling that is seen to accompany acute or even chronic Bright's disease. The subsidence of dropsy is necessarily accompanied by an increased flow of urine, and very commonly the increase in the urinary flow occurs before there is any obvious diminution in the amount of dropsy.

The fluid of renal dropsy is poor in proteids and rich in salts and nitrogenous extractives, and in the case of the subcutaneous fluid in general renal anasarca the proportion of proteid present may be less than 1 per cent. The fluid in the peritoneum and serous cavities generally is richer in proteid, but never contains the quantities seen in the cardiac dropsy. The proteids present in the blood in cases of Bright's disease, especially in chronic Bright's disease, may fall to as low as 5 per cent.

The dropsy of renal disease affects especially the subcutaneous tissue, and may reach a high degree of development in this without any large amount of fluid being poured out in the serous cavities. In some instances the dropsy of renal disease is more marked in the face, in others in the legs, or in the scrotum, sacrum, etc. It is especially liable to vary in its amount in different parts according to

the posture of the patient. In severe cases the effusion not only involves the serous cavities, but also some of the solid organs, especially the lungs, and to a less extent the brain and larynx. Œdema of the lungs is one of the most formidable complications of Bright's disease, and is especially apt to occur in uræmia. Ascites and hydrothorax are unlikely to be present unless general anasarca also exists.

The dropsy of renal disease is perhaps dependent on some alteration in the composition of the blood or the presence of some toxic substance in the general blood stream, which damages the walls of the vessels, allowing the fluids of the plasma to pass out. Some writers have thought that it is dependent simply on a condition of hydræmic plethora, this being brought about as a result of the scanty secretion of urine. For the reasons mentioned it is difficult to regard renal dropsy as dependent simply on the suppression of the renal functions, and it is more probable that the scanty secretion of urine accompanying the onset of the dropsy is the result of the latter rather than its cause.

Very violent acute nephritis may be seen sometimes as a result of the action of toxic substances like turpentine, or occurring in the course of such a malady as diabetes without the occurrence of dropsy, and without any diminution in the quantity of urine excreted (see also Vol. IV. pp. 464, 474).

Disorders of nutrition.—In most renal maladies emaciation is present, but in some it is a marked feature, and may rival in amount that seen in malignant disease. The amount of waste is often concealed by the presence of dropsy, and is most obvious in those forms which are not accompanied by this condition, *e.g.* granular kidney, cystic kidneys, etc. The wasting is in part dependent on the serious gastric disturbance so often present in renal disease leading to loss of appetite, nausea, vomiting, but it may be in part associated with the destruction of the kidney substance, and comparable with the experimental results described above (p. 563).

Diminished resistance.—Patients suffering from certain renal affections, more especially Bright's disease, are prone to suffer from a variety of so-called complications, such as pericarditis, pneumonia, pleurisy, peritonitis, etc. In some of these instances, especially in pericarditis, the effusion has been found to be sterile, but in the majority of cases the secondary inflammations are dependent on the occurrence of microbic infection, and it has long been known that such patients are very unsuitable for surgical operations owing to the tendency to septic complications unless the most rigid antiseptic

precautions are taken. Incisions made for the relief of dropsy in renal disease are very liable to become infected, and erysipelas and other septic complications are liable to develop. In all probability these phenomena are, in part, dependent on a diminished resistance of the tissues to microbic infection.

Pain in diseases of the kidney.—Pain is a very variable symptom in diseases of the kidney. Many of the most serious affections of this organ run their whole course without the production at any time of pain. In others it may be almost the only symptom present. When this is so the pain may be of the variety known as referred pain, and is especially associated with diseases of the pelvis of the kidney and of the ureter. In affections of the kidney substance itself pain vaguely localised in the loin, and of a dull aching character, may be present. A typical instance of this is afforded by cystic disease of the kidneys. A somewhat similar pain, also vaguely localised in the loin, may be experienced in acute congestion and inflammation, but Bright's disease and other destructive diseases of the kidney do not usually give rise to pain. Infarction not uncommonly gives rise to sudden pain, which is usually described as lumbar in position, but instances are not rare where such pain is experienced in the abdomen. It is probable that in all affections limited to the kidney substance the pain is felt in some part of the area supplied by the tenth dorsal segment, and may therefore reach as far forward as the umbilicus and middle line. In some gross diseases of the kidney, *e.g.* abscess, pyonephrosis, movable kidney, tenderness may be elicited by pressure on the organ, and it is not rare for a peculiar sickening sensation to be experienced when the kidney is grasped between the two hands in bimanual palpation. In disease of the pelvis of the kidney, especially calculus, tubercle, bilharzia, pain may also be experienced in the area of the tenth dorsal, but frequently also involving that of the eleventh. It would seem that if the calculous or other disease remains limited to the pelvis, that the pain does not radiate into the groin, but remains confined to the loin. On the other hand, in affections of the ureter the eleventh and twelfth dorsal are the segments to which the pain is referred, and it therefore extends to the groin, scrotum, and thigh. In exceptional instances it has been stated that the pain of renal calculus may extend down the leg even to the sole of the foot. The pain in the loin is often constant, whereas that in the groin is frequently paroxysmal. Attacks of pain, almost indistinguishable from those of renal colic, occur sometimes in movable kidney, and in very exceptional instances similar attacks of pain have developed

without any gross lesion or preternatural mobility of the organ being found on surgical exploration. It is not uncommon for these attacks of pain simulating renal colic to cease after an exploratory operation, and it is possible that they may be dependent on slight kinking of the ureter or of the renal vein, giving rise to distension or engorgement of the organ.

SPECIAL SYMPTOMATOLOGY AND TREATMENT OF RENAL DISEASES

DISPLACEMENTS OF THE KIDNEY

Symptoms.—Many of the displacements and of the anomalies of the kidney do not give rise to symptoms, and are only discovered in the post-mortem room. This is generally the case with horse-shoe kidneys. In some instances of fixed displacements of the kidney kinking of the vein may occur, and in this way considerable swelling of the organ is produced. If the displaced organ be situated in the iliac fossa the tumour produced by the swollen kidney may be erroneously diagnosed as due to appendicitis. Errors of diagnosis also may occur when the kidney lies in the hollow of the sacrum, and it may be impossible in such cases to avoid mistakes.

Movable kidney is by far the most important of the displacements of the organ. Movable kidney is very rare in men, and occurs at least seven times more frequently in women. It is also most common on the right side, but both organs may be affected. Laxity of the abdominal walls and emaciation, especially the former, are the most important predisposing causes. The laxity of the abdominal walls may be the sequel of pregnancy, but the affection is frequently seen in childless women. Tight lacing has been considered by some writers as an exciting cause, by others as only predisposing to the condition. In a few instances mobility of the organ has resulted from injury, sometimes direct to the loins, more often indirect, *e.g.* jumping. Movable kidney is common in neurasthenics, and the laxity of the abdominal muscles may be dependent on mere loss of tone. In many instances of slight mobility no symptoms are produced, and in a small proportion the symptoms produced are very severe. In the great majority of cases they are of an intermediate degree of severity. The most constant symptom is pain, and this is usually felt in the abdomen

and loin, and is associated with tenderness of the kidney on palpation. The pain may be accompanied by nausea and vomiting, and in some instances it may resemble that of renal colic in its distribution. A constant aching in the affected loin is not uncommon, and then attacks of more severe pain, accompanied by nausea and vomiting, occur from time to time. The pain may be accompanied not only by abdominal tenderness, but also by distension. Hydronephrosis is a comparatively common accompaniment of movable kidney, and it is occasionally intermittent in character. Hæmaturia, dependent on kinking of the renal vein, may also occur. Movable kidney is sometimes associated with dilatation of the stomach, and even with the presence of slight jaundice, both these effects being dependent on the traction on the pylorus and bile duct produced by the displaced kidney. Excessive and distressing pulsation of the aorta is another very common coincident manifestation. In the more severe forms of movable kidney extreme neurasthenia may be present, and the patient often seeks advice rather on account of this than on account of the abdominal symptoms.

Where gastric symptoms are marked the existence of movable kidney may be overlooked, all the patient's troubles being put down to the stomach condition. Too much stress may be laid on the presence of movable kidney as a cause of neurasthenia, and in many such patients cure of the renal condition does not lead to the cure of the neurasthenia. In cases where hæmaturia is present movable kidney may be confounded with renal colic. The condition is actually diagnosed by feeling the kidney come down during inspiration, and by the possibility of grasping the displaced organ between the hands and keeping it in the displaced condition.

Prognosis.—The condition is not serious in itself, but when associated with neurasthenia of a severe type the prospect of cure or relief is not always good. Further, movable kidney may at any time become complicated by the development of hydronephrosis, and this complication, owing to the presence of severe pain, may lead to considerable impairment of the general health.

In the majority of cases no **treatment** is required, more especially if the condition produce no symptoms, and certainly the mere existence of a slight degree of mobility of the kidney in cases of neurasthenia does not call for any special attention. In those cases where definite symptoms are produced much may be done by improving the general nutrition so as to favour the deposition of fat, and in some instances Weir Mitchell treatment is useful. This may also be beneficial by tending to increase the tone of the

abdominal muscles. A properly fitted pad, to be put on before the patient assumes the erect posture, may often give great relief, and such pad is much oftener of benefit when made and fitted by the patient than are the elaborate belts of the instrument makers. If the symptoms are very severe, and none of these measures procure relief, and more especially if such a complication as hydronephrosis be present, surgical measures, such as fixation of the kidney, must be employed. In a very small proportion of cases, especially where hydronephrosis has been produced, and where fixation may have failed, nephrectomy may be necessary.

PASSIVE CONGESTION OF THE KIDNEY

Symptoms.—In passive congestion of the kidney a scanty urine is secreted, amounting often to less than twenty ounces per diem. The specific gravity is usually above the normal, and the urine is high-coloured and tends to deposit urates abundantly. Albumin in variable quantity is present, not usually exceeding one-third, although the mere presence of larger amounts of albumin does not negative the existence of passive congestion. Blood may be present, but is not usually abundant, and frequently occurs only in the form of blood casts. Hyaline and granular casts are also seen. In many cases dropsy is a marked symptom, but, when present, it is of cardiac, not of renal type.

Diagnosis. — Passive congestion of the kidney has to be diagnosed from active congestion and from Bright's disease, and this is often difficult, if not impossible, if the opinion be based simply on an examination of the urine. In nephritis and in Bright's disease casts are generally more abundant, and contain definite renal elements, and the urine is not usually of high specific gravity nor of high colour. Speaking generally, the amount of albumin is greater in cases of nephritis than in cases of passive congestion, but this is not invariable. The existence of Bright's disease may often be detected owing to the secondary changes in the vessels, as shown by the state of the pulse and the appearance of the fundus oculi. The difficulty of separating congestion of the kidney from Bright's disease arises chiefly in cardiac cases.

The **prognosis** of the condition depends mainly on its cause. As a rule, the existence of passive congestion of the kidney increases the gravity of cases of heart disease, but the prognosis

is far better than in cases of heart disease complicated by Bright's disease.

The **treatment**, like the prognosis, depends essentially on the cause. All the measures employed are directed to improve the efficiency of the circulation through the kidney, thus digitalis, caffein, strophanthus, are all of value, but in the majority of cases digitalis is the most useful drug, or a combination of digitalis and caffein. The latter is of value where an immediate and sudden effect is required, but the drug soon loses its power, as it has not the persistent action seen with digitalis. Free purgation, especially with saline cathartics, is also of great value.

The diet should be restricted to milk only. Stimulants may be required, owing to the cardiac disease causing the renal congestion, but they should be given sparingly, and the patient encouraged to take simple diuretic drinks.

NEPHRITIS

Symptoms and diagnosis.—As already mentioned, it is exceedingly difficult to separate active congestion, nephritis, and the slighter forms of acute Bright's disease, as they all merge into one another. The term nephritis is commonly employed to describe the slighter forms of acute congestion, and also the changes that occur in the kidneys during the height of the acute specifics. In a very large proportion of cases of nephritis there are no symptoms in the true sense of the word, and the existence of the complication can only be diagnosed by the state of the urine. This is diminished in quantity, and in cases of severe nephritis complete suppression may occur. Blood is usually present, and in the graver forms of the disease it may be present in large quantity. Albumin is invariably present in greater or less amount, sometimes little more than a trace, in others the urine may be almost solid. Hyaline, granular, epithelial, and blood casts may all be present.

Pain in the back may be present in the more severe forms of acute nephritis, and more especially when nephritis arises as a result of renal embolism. In this condition the pain may be felt in the back and in the loin, and is usually quite sudden in its onset. The urinary changes seen in infarction of the kidney are also quite sudden. In the majority of cases albuminuria suddenly appears, and, after persisting for a few days, disappears with equal suddenness. In exceptional cases the onset of infarction is marked by the occurrence of profuse hæmaturia, which may after a few

days subside completely. It is important to recognise that in infarction quite considerable quantities of blood and albumin may suddenly appear in the urine, and it is for this reason that the condition may be confounded with a severe and general nephritis. Infarction is never accompanied by dropsy.

Nephritis and acute congestion have to be separated, on the one hand, from passive congestion, and, on the other, from the slighter forms of acute Bright's disease. It is distinguished from passive congestion partly by the characters of the urine, especially the presence of epithelial casts, and partly by the circumstances under which the complication arises—the one occurring as an accompaniment to cardiac and lung diseases, the other as a result of the action of toxic substances or the presence of an acute febrile disorder. In cardiac cases the possibility of infarction must be always considered, the mere occurrence of a sudden albuminuria or hæmaturia by no means necessarily indicating the presence of simple active or passive congestion. It is important to recognise that in many instances of infarction hæmaturia does not occur. It is practically impossible to draw a distinction between the severer forms of nephritis and the slighter forms of Bright's disease, and there is much to be said for the view that they are really the same. The severer forms of acute Bright's disease are at once distinguished from nephritis by the occurrence of dropsy in the former.

The **prognosis** of nephritis is usually good, especially of that form which occurs during the height of the incidence of acute specifics. Where it is dependent on the toxic action of turpentine or cantharides the case becomes one indistinguishable from acute Bright's disease, and the prognosis may be more serious. Very severe forms of acute nephritis sometimes occur during the height of acute specifics, such as scarlet fever, and may be accompanied by suppression, and cause death from uræmia. Renal infarction is not usually followed by serious consequences unless the infarct be septic, as in cases of malignant endocarditis. Under such circumstances renal abscess or perinephritic abscess may be produced.

Although acute nephritis may completely subside and the urine regain its normal characters, it is probable that kidneys affected by this disease may be more liable to undergo the changes of chronic Bright's disease subsequently, although the transition from the one to the other is not direct nor immediate, an interval of many years elapsing between the two conditions.

The **treatment** of nephritis necessarily merges into that of the

underlying acute disease so often causing it. In all cases it is very essential that simple unirritating food should be ordered, and in most cases a pure milk diet is the most satisfactory. Diluents should be freely given, and in the more severe forms of nephritis, where suppression of urine and uræmic symptoms occur, free purgation is necessary, and the activity of the skin should be promoted by hot-air baths or the wet pack.

BRIGHT'S DISEASE

ACUTE BRIGHT'S DISEASE.—The initial **symptoms** of acute Bright's disease vary considerably in their severity. In exceptional instances the disease is ushered in with rigor, fever, and the secretion of a scanty blood-stained urine. Pain in the back may be present, but is not usually a marked symptom. In such cases as the above, dropsy, involving the subcutaneous tissue of the body generally, may also occur, and the severity of this is sometimes such that in the course of twenty-four to forty-eight hours the patient may become so bloated as to become almost unrecognisable. On the whole, it is unusual even for acute Bright's disease to commence with such marked symptoms. A more usual mode of onset is for the patient to present indefinite signs of illness, such as slight shivering, pains in the limbs generally, nausea, and lassitude. The urine is then noticed to contain blood, and on examination is found loaded with albumin. In other instances the first sign of illness is the occurrence of dropsy in the face, scrotum, or limbs.

The changes in the *urine*, more especially the presence of blood, may be the only signs that attract attention to this serious disease; many patients think that the symptoms are simply those of a severe cold. There are considerable variations as to the extent to which *anasarca* is developed in acute Bright's disease. Very commonly it is moderate in amount, and it does not generally reach the high degree of development seen in chronic Bright's disease. Dropsy may be completely absent, and this is sometimes the case in those instances where the urine contains large quantities of blood. In the most severe forms of acute Bright's disease the diminution in the amount of urine may be such that actual suppression occurs. It is more usual for the quantity excreted to vary between some ten to twenty ounces. *Anæmia* develops rapidly in most cases, and this, together with the presence of the dropsy, causes the character-

istic pale and pasty appearance of the patients. The mucous membranes, such as the conjunctivæ and the lips, are usually pale. *Hæmorrhages* from the nose and purpuric extravasations into the subcutaneous tissue are not uncommon. *Dyspnœa* is often a marked symptom, dependent partly on the anæmia and partly on the dropsical accumulations in the peritoneal and pleural cavities; it may, however, be of cerebral origin, and due to uræmia. *Gastro-intestinal* symptoms are almost always present, and often reach a high degree of severity. Nausea, retching, hiccough, vomiting, all occur, and nausea is especially apt to be severe. The state of the bowels varies considerably, but diarrhœa may be present, and when this is the case it is often severe in amount. Stomatitis and hæmorrhages beneath the mucous membrane of the mouth are common in the more severe forms of the disease, and this in part leads to the fœtor of the breath so often noticeable. The tongue is apt to become dry, brown, and cracked, and the patients frequently complain greatly of an extremely foul and unpleasant taste in the mouth. *Fever* is by no means a conspicuous symptom in acute Bright's disease; very commonly the temperature is but little raised above the normal even at the onset, and subsequently to this it may even be subnormal. In quite exceptional instances the disease is ushered in by fever, amounting to 102° or even 104°.

Uræmic symptoms are usually present to a varying degree in all instances of acute Bright's disease; thus the nausea, vomiting, etc. described above are, in all probability, strictly speaking, of uræmic origin. The more serious uræmic symptoms, such as drowsiness, delirium, mania, coma, convulsions, catalepsy, urgent dyspnœa, amaurosis, twitchings, and cramps, together with cutaneous eruptions of a papular, erythematous, or bullous type, are only seen in the more severe forms of the disease.

The *cardio-vascular system* is affected very early in acute Bright's disease, and in the course of a few weeks, from three to six, the heart may show definite signs of enlargement, and the ordinary characteristics of a high-tension pulse may be quite well marked. Cardiac dilatation may also occur even at an earlier period, and this formidable complication may be the cause of death after an illness of a duration of two or three weeks, or even less.

A considerable number of **complications** may occur during the course of acute Bright's disease, more especially inflammation of the serous membranes or of the lungs. Pericarditis, peritonitis, and pleurisy are all serious, especially the first. Pneumonia is also a frequent complication, and, in cases where uræmic symptoms are

marked, pulmonary œdema may supervene and cause death even where general dropsy is absent or slight.

Retinal changes, such as papillitis and albuminuric retinitis, are more characteristic of chronic Bright's disease than of the acute variety, but still these lesions are seen occasionally to arise during the course of the acute disease.

Course and termination.—In the great majority of cases the symptoms of the acute disease gradually subside, the amount of blood in the urine diminishes, and the quantity of urine increases, until the amount excreted considerably exceeds the normal. This increased excretion is especially marked where dropsy has been present and is subsiding, but may occur to a less extent where general anasarca is, at any rate, not obvious. The urine may remain pale and of low specific gravity for a considerable time after it ceases to be albuminous.

Wasting, which is a prominent sign of Bright's disease, becomes especially noticeable during convalescence, after the subsidence of the acute symptoms.

In some instances although the symptoms subside they do not disappear, and the patient passes gradually into the condition characteristic of chronic Bright's disease.

The mortality of acute Bright's disease is considerable, and the severe cases occurring as a result of scarlet fever are especially liable to be fatal.

Chronic Bright's disease.—The onset of chronic Bright's disease is frequently extremely insidious, so that, in a large proportion of cases, the patient is quite unable to date the beginning of the malady. Some marked sign, such as general dropsy or greatly increasing pallor, is noticed to occur after a period of vague ill-health. In a small proportion of cases the symptoms of acute Bright's disease merge into those of the chronic disorder, but it is probable that the great majority of cases of chronic Bright's disease are really chronic from the beginning, and not the direct sequel of the acute condition. The malady, when resulting from the action of mineral poisons or alcohol, is generally of this primarily chronic form. Even where the malady is associated with acute Bright's disease it frequently happens that the initial attack of the acute disorder completely subsides, and the urine regains its normal characters. Then the patient suffers from a second attack, and after a variable period this may pass into a condition of chronic Bright's disease, or it also may subside, to be followed by a third or subsequent attack, which becomes chronic.

The **symptoms** of chronic Bright's disease depend a good deal on the variety of the lesion present. *In the first form*, the so-called large white kidney, the *urine* is scanty, pale, and highly albuminous, and contains abundant casts, and the patient usually suffers from considerable dropsy, not only of the subcutaneous tissues, but also of the serous cavities. The *anasarca* may reach a very high degree of development, and *anæmia* is a marked feature. *Stomatitis* and submucous *hæmorrhages* in the mouth often appear, and the *pulse*, although showing signs of increased tension, does not present these to a very great extent. *Gastric symptoms*, especially nausea and vomiting, are usual; diarrhœa may be very troublesome and, in some instances, associated with the presence of ulcerative colitis. *Pericarditis*, *pleurisy*, and *pneumonia* are all liable to occur, and the first is especially prone to run a latent course.

In the more severe forms of this variety of chronic Bright's disease death may occur from general asthenia, or from the embarrassment of the circulation and respiration dependent on the dropsy. Subacute and chronic uræmia are often present, and in exceptional cases death may occur from cerebral hæmorrhage. Secondary inflammations are especially liable to cause death when associated with uræmia. Although the disease is so very serious and the ultimate outlook so grave, patients may live for many years after the occurrence of general dropsy, and even after the first appearance of marked uræmic symptoms. Both the general dropsy and the uræmic symptoms may sometimes subside in a remarkable manner and the patient return to a moderate degree of health, notwithstanding the fact that the urine remains highly albuminous and presents the features characteristic of chronic Bright's disease.

In the *second variety* of chronic Bright's disease, described above as the contracted white kidney, the symptoms are quite different. In such patients *dropsy* is not a marked symptom, and it is commonly completely absent and in many cases has never been present at any time. The quantity of *urine* excreted is notably increased, and it contains a considerable amount of albumin, often as much as one-third, and such patients usually come under observation complaining either of uræmic symptoms, or else on account of pallor and *anæmia* and the occurrence of great loss of strength, together with marked wasting. *Headache* is often a prominent symptom, and one for which advice is sought. Many of these patients have a peculiar *earthy complexion*, and owing to the marked wasting they may present a superficial resemblance to the cachexia seen in malignant disease. *Pigmentation* of the skin may

sometimes occur, and instances are on record where the malady has been confounded with Addison's disease. In exceptional instances profuse *hæmaturia* may set in, simulating in its abundance that seen in malignant disease of the kidney or in renal calculus.

Uræmia is especially prone to occur in this form of Bright's disease, and it may do so with great suddenness and the symptoms be exceptionally violent. In some instances acute and fulminating uræmia has occurred at a time when there were no obvious symptoms pointing to the underlying grave condition. It is more usual, however, for acute uræmia to supervene after the malady has advanced to a sufficient extent to produce obvious ill-health. In some instances the occurrence of uræmia is determined by the onset of some intercurrent illness, such as influenza, pneumonia, etc. Cases are on record where the occurrence of severe epileptiform seizures in a patient apparently well has been the first indication pointing to the existence of the chronic Bright's disease.

The *cardio-vascular system* shows all the characteristic evidences of high tension, and examination of the fundus oculi reveals the presence of papillitis and neuro-retinitis.

The **diagnosis** of acute Bright's disease is usually easy, owing to the occurrence of dropsy together with the changes in the urine. It may have to be distinguished from transitory nephritis, from renal infarction, and also from passive congestion. The last two conditions are especially apt to occur in cases of cardiac disease, and the dropsy dependent on the cardiac lesion may be erroneously thought to be of renal origin. In the nephritis accompanying the acute specifics dropsy is absent, and the urinary changes are not usually so marked as those seen in acute Bright's disease. Still, the difference between the two conditions is largely one of degree, as dropsy is not an invariable accompaniment of acute or even of fatal Bright's disease. In renal infarction the effects are not only produced much more suddenly than in acute Bright's disease, but the urine recovers its normal characters much more rapidly. In the severe forms of passive congestion associated with morbus cordis blood may be present in the urine in considerable abundance, and hence the urine may present a certain resemblance to that seen in acute Bright's disease. In the latter, however, casts are abundant, and it is more usual for the blood to be altered, whereas in renal congestion, even if the hæmorrhage be profuse, the urine still remains of a red colour. In very exceptional cases it is said that acute nephritis may occur with dropsy, but without the presence of albumin or blood in the urine.

Lardaceous kidney may be confounded with chronic Bright's disease, as in both the amount of albumin may be large. The differential diagnosis can usually be arrived at by the detection of the presence of the morbid change in other organs, *e.g.* the liver and spleen, and by the absence of the involvement of the cardio-vascular system in lardaceous disease.

Difficulties sometimes present themselves in differentiating between the slighter forms of chronic Bright's disease and so-called physiological albuminuria. In both of these conditions the amount of albumin in the urine may vary as a result of changes of posture, and it is often difficult to decide with certainty whether slight albuminuria of this character, sometimes seen after an attack of acute nephritis, is to be looked upon as evidence of a progressive lesion or not. The examination of the heart, the vessels, and the fundus oculi will often assist greatly in determining whether albuminuria is or is not dependent on the presence of serious renal disease.

In the second variety of chronic Bright's disease, where dropsy is absent and the symptoms insidious, the condition may be overlooked, especially if the urine be not examined. The severe headache and vomiting may lead to the diagnosis of cerebral tumour if the urine be not examined, or the uræmic seizures may be thought to be of epileptic origin. The marked wasting that is often a prominent feature may lead to the suspicion of the existence of malignant disease, and pigmentation of the skin is sometimes very marked and may cause confusion with Addison's disease. Careful examination of the urine in all cases will prevent most of these errors, although in very exceptional instances fatal chronic Bright's disease may occur where albumin is either completely absent or only present in traces.

The **prognosis** of Bright's disease, both acute and chronic, depends a good deal on the cause. Thus the acute form seen as a sequel of scarlet fever is not uncommonly fatal. The secretion of a very scanty urine and the early prominence of uræmic symptoms are unfavourable signs, as is also the occurrence of cardiac dilatation, accompanied by a very frequent pulse. Recovery, however, may take place after the occurrence of severe uræmic seizures, and even in cases where for a time there is almost complete suppression of urine. Acute uræmia occurring in acute Bright's disease is, on the whole, not so unfavourable a symptom as acute uræmia in chronic Bright's disease. In a considerable proportion of cases the symptoms subside, and in the course of a few weeks the dropsy

and then the albuminuria disappear. In some instances, although the more severe symptoms subside they do not disappear, and the albuminuria persists so that in the course of some months a condition of chronic Bright's disease is established. Where the albuminuria has persisted for six months, it is usually stated that it is exceptional for it ever to disappear, but this statement is by no means universally true, and cases are on record where after continuing for some years the albuminuria has gradually entirely disappeared. In some cases neither the albuminuria nor the dropsy subsides entirely, although undergoing some improvement, and the disease is ultimately fatal in the course of a few months from the onset. Even in these cases, however, the progress of the malady is rarely uniform.

In cases of chronic Bright's disease, whether the sequel of acute Bright's or chronic *ab initio*, the outlook is generally unfavourable, and the duration of life very uncertain, although some such patients may live for five, ten, or even twenty years, passing large quantities of albumin all the time. The available data for accurate prognosis are however scanty, owing to the great risk there is in all these cases of the occurrence of sudden complications. Thus cerebral hæmorrhage may occur at almost any moment in both varieties of chronic Bright's disease, and it is often impossible to foresee its imminence, although the presence of high tension and evidence of arterial degeneration and cardiac hypertrophy should cause a guarded forecast. Intercurrent illnesses of a more or less trivial character may, in a person suffering from chronic Bright's disease, lead to death from uræmia, and uræmia may also arise as a result of indiscretions in diet. For all these reasons the prognosis is most uncertain. In cases where dropsy is absent and the cardio-vascular changes are moderate in amount, and there is no marked wasting, the patients may live in a condition of moderate health for many years, notwithstanding the existence of marked albuminuria. Speaking generally, the prognosis in the contracted form of Bright's disease, where marked cardio-vascular changes are present and dropsy absent, is more unfavourable than in the other variety. Such patients often die with extreme suddenness only a few days or weeks after the onset of the symptoms causing them to seek advice. The presence of albuminuric retinitis is also unfavourable, and although many such patients may succumb a few weeks or months after coming under observation, yet life is sometimes maintained for several years after the detection of this complication.

Treatment.—It is essential in the treatment of Bright's disease

to diminish as far as possible the work of the kidney, and to promote the free action of the skin and of the bowels. The nitrogenous constituents of the food should be diminished as far as practicable, and during the acute stage the diet should be as small in amount as possible. During the acute disease milk only should be given, and this in quantities of from one to three pints in the twenty-four hours. Smaller quantities will suffice for a short period during the most acute stage of the malady, although such quantities are not sufficient to maintain nitrogenous equilibrium. The milk should be freely diluted with water, soda-water, lime-water, or gruel. If, as is often the case, vomiting be a marked symptom the milk should be pancreatised and given in small quantities. Barley water and gruel may also be used, but soups, meat extracts, or beef-tea should be avoided, both on account of their small nutritive value and also owing to the large quantity of extractives they contain. During the acute stage the quantity of food that is actually absorbed is often very small, owing to the vomiting so frequently present. Rectal feeding may be necessary, and the nutrient enemata used should consist of pancreatised milk or milk and gruel.

Milk is not only useful as an exclusive diet in acute Bright's, but it may also be used with great advantage in the treatment of chronic Bright's disease, especially where uræmic symptoms are present or where dropsy is marked. In the chronic form of the disease large quantities of milk are required, as at least from three to four pints must be taken by an adult per diem in order to maintain, even approximately, nitrogenous equilibrium. Owing to the albuminuria considerable quantities of proteid are lost, and this loss must be met as far as possible from the proteid of the food. It is no uncommon thing for the patient suffering from chronic Bright's disease to pass daily in the urine quantities of proteid equal to the amount present in from one to two pints of milk. A milk diet may be persisted in in the treatment of chronic Bright's disease for weeks, months, or even years, but it is of most use in the more severe forms of chronic Bright's disease, and in cases of subacute exacerbation of the chronic disease. Not uncommonly the general nutrition of the patient is not sufficiently well maintained on a milk diet, and in these cases it is unwise to persist in it. Additions in the form of bread, fish, chicken are frequently called for, and in cases of chronic Bright's disease where uræmic symptoms and dropsy are absent, an ordinary diet of meat and vegetables may often not only not increase the albuminuria, but may lead to such an improvement in the general health as to justify

its being given. In all cases it is essential that highly seasoned articles of diet should be avoided, and that all food should be plainly cooked. Meat, if allowed, should be taken in small quantities, probably not oftener than once a day.

In all cases of chronic Bright's disease where uræmic symptoms are present, even when slight in amount, or where dropsy occurs, the patient should be put to bed and kept on a rigid milk diet until the urgency of these symptoms has subsided.

Stimulants are contra-indicated in all forms of Bright's disease, acute and chronic, owing to their irritating action on the kidney. It may, however, be necessary if circulatory weakness be marked, to order stimulants, but they should be always prescribed in small amounts and freely diluted. It is usually held that they are best ordered in the form of whisky or gin.

Diluents, such as simple mineral or alkaline waters, may be partaken of freely, and hot drinks are often useful in combination with hot-air baths for the purpose of promoting sweating. The activity of the skin should be promoted by all available means, such as the wet pack, hot-air baths, and not infrequently sweating produced by pilocarpine will afford relief in the treatment of uræmia. The hot-air bath, although extremely useful, must be used with caution, as in some instances there is a tendency to syncope, and more particularly on its first employment. Uræmia has also occasionally apparently been induced by the use of the hot-air bath, but this is so exceptional that it is not a valid reason against its use. The temperature of the hot-air bath may vary from 120° F. to 180° F., but the higher temperatures should not be used until the patient have got accustomed to such baths.

Free purgation, especially with saline purgatives, is an essential point in the treatment of both acute and chronic Bright's disease, as there seems little doubt that large quantities of extractives and possibly of toxic materials may be eliminated by the alimentary canal. Although such a course is advisable, it is very necessary to avoid causing profuse diarrhœa by the injudicious administration of purgatives. Enteritis and even ulcerative colitis may occur as complications of Bright's disease, and it is possible that these conditions may be, if not started, at any rate aggravated by the administration of too violent purgatives.

Hot applications to the loins or dry cupping may be useful to relieve the pain in the back that is sometimes experienced in acute Bright's disease.

The dropsy must be treated on the general lines mentioned

above, more especially by purgation. It, however, rarely undergoes any marked alteration until the condition of the kidney has improved. In exceptional instances, where the amount of anasarca is very great, incisions may be necessary in order to relieve the general swelling. In such cases the utmost precautions must be taken to avoid the possibility of septic infection, as such complications follow a rapid and most unfavourable course in persons suffering from renal disease. Accumulations of fluid in the thorax must be removed by paracentesis.

Uræmia must also be treated by the general measures directed to improve the condition of the kidneys, by promoting elimination by the skin and bowel, but in addition and in the more severe forms venesection may be useful, or venesection followed by transfusion; the latter by itself is necessarily of no avail. Chloroform and morphia may be used in the treatment of uræmic convulsions, and morphia is useful for the relief of other uræmic symptoms, *e.g.* dyspnœa. The administration of morphia, however, in renal disease is not free from risk, especially in cases where the quantity of urine is very greatly diminished, and where there is respiratory embarrassment. At the same time the dangers of morphia in renal disease have undoubtedly been exaggerated.

The cardiac weakness sometimes developed in the course of Bright's disease as a result of the onset of cardic dilatation should be treated by the administration of digitalis and strychnine, notwithstanding that there are theoretical objections to the use of digitalis in this condition, owing to the fact that it increases the blood pressure, and it may be argued that the heart is already failing as the result of increased pressure. It is probable, however, that in many cases the cardiac failure is not immediately dependent on the increased blood pressure, but rather on primary effects produced on the heart muscle. However this may be, there can be little question but that both in acute and chronic Bright's disease digitalis is often of considerable value, as it not only may produce an improvement in the force and rhythm of the heart's action, but it may also lead to an increased secretion of urinary water as a result of the general improvement in the circulation.

The anæmia of Bright's disease, which is often extremely marked, should be treated on the usual lines by the administration of iron and of arsenic, the combination of the two or the latter alone is often more useful than iron by itself.

Patients who have suffered from acute Bright's disease should subsequently winter in a warm climate, and patients suffering from

the chronic form of the malady should, if possible, winter abroad, in such places as Egypt, Algiers, West Indies, Madeira.

LARDACEOUS DISEASE OF THE KIDNEY

The **symptoms** seen in cases of waxy diseases of the kidney are essentially similar to those occurring in chronic Bright's disease, but, except in the terminal stages of the malady, are not of the same degree of severity; hence it is less common for patients to come under observation for the symptoms produced by the renal affection, especially as the malady is invariably seen as a complication of other and graver disorders.

Anæmia is a marked symptom, and in the later stages *dropsy* also occurs similar in distribution to that seen in Bright's disease. In the earlier stages dropsy is absent, and the patient secretes an abundant dilute urine containing only small quantities of albumin. Where dropsy is marked the quantity of urine necessarily undergoes diminution, and the percentage of albumin is considerably higher. In all instances where the degeneration is extensive some uræmic symptoms are present, and death may occur from this complication. This is sometimes the case after exploration of the kidney by operation, *e.g.* where lardaceous degeneration complicates pyelitis. Hyaline *casts* are usually present in the urine, and sometimes casts that have undergone waxy degeneration are also found.

Diagnosis.—The disease is usually distinguished from chronic Bright's disease by the absence of the involvement of the cardio-valvular system, and by the presence of some underlying disease such as syphilis, tubercle, or prolonged suppuration, in fact any of the recognised causes of amyloid degeneration. Further evidence of the disease in other organs, such as the liver, spleen, and intestines, is often to be obtained.

The **prognosis** of the affection depends almost entirely on that of its cause. If this can be successfully removed great improvement, if not cure, may ensue, provided the disease be not too far advanced.

Treatment.—In the more severe forms of the affection where dropsy and uræmic symptoms are present the malady must be treated in the same manner as Bright's disease. In the slighter forms, however, and in the earlier stages, where the condition is only revealed by the presence of albuminuria, a much more liberal diet than that given in Bright's disease should be ordered.

This is necessary owing to the malady occurring as a complication of other debilitating diseases, and also because in the early stages the excretory activity of the kidney is by no means seriously impaired. Tonics and stimulants are also frequently required as well as good food.

GRANULAR KIDNEY

The **symptoms** of patients suffering from granular kidney present considerable variety. In some instances they are essentially cardiac, in others the first effects noticed are failure in the nutrition of the body. In a third group uræmic symptoms occur, and in a fourth the first serious symptoms produced are those resulting from cerebral hæmorrhage.

In the *cardiac group* the patients come under observation suffering from shortness of breath, palpitation, and not uncommonly from giddiness, especially on change of posture. The palpitation is often accompanied by a throbbing headache, and this is marked in all varieties of granular kidney, and sometimes is of such severity as to be the leading symptom for which the patient seeks advice. Cardiac pain, which is usually restricted to the precordial area, but may have a distribution somewhat similar to that seen in the slighter forms of angina pectoris, is a frequent concomitant. The cardiac symptoms of pain, palpitation, and dyspnœa may reach a high degree of development, and may be associated with a considerable degree of cardiac hypertrophy and dilatation without the necessary occurrence of any dropsy. In other instances dropsy is a more marked feature, and has very much the distribution and characters of that seen in mitral regurgitation. Such cases clinically are very apt to be mistaken for cases of simple mitral regurgitation, whereas in reality the mitral regurgitation depends on cardiac dilatation supervening as a result of failure of the hypertrophied heart accompanying the granular kidney. In most instances these cases may be readily diagnosed owing to the increased tension of the pulse and the thickening of the arteries. In some cases, however, the cardiac failure is so great in amount as to obscure the character of high tension in the pulse.

Many cases of granular kidney come under observation owing to a *progressive failure in nutrition*, loss of appetite, of strength, and of flesh, and in a certain proportion the wasting is very considerable in amount, almost rivalling that seen in some forms of malignant disease. The skin often has an earthy and somewhat cachectic

appearance, and a moderate amount of pigmentation is not uncommon. Examination of the pulse and of the urine usually reveals the real nature of the case.

Uræmic symptoms of varying degrees of severity are frequently met with, more especially nausea, vomiting, and cramps with twitchings of the extremities. Exceptionally, more violent forms of uræmia may apparently usher in the malady, in other words, the disease is latent until the uræmia declares itself. In such instances the uræmic phenomena may be of a maniacal, cataleptic, or epileptiform type, or even simple severe dyspnœa simulating asthma. In some of these cases the onset of the uræmic symptoms is associated with the development of complications, such as pericarditis, a condition of fair health having existed up to the time of the appearance of the complication.

Granular kidney may run a latent course until the development of some serious and perhaps fatal complication, such as pericarditis or cerebral hæmorrhage. *Cerebral hæmorrhage* is especially apt to be very extensive and often rapidly fatal, owing to the fact that the arterial degeneration leading to the hæmorrhage is accompanied by a condition of high tension.

The *urine* in cases of granular kidney is secreted in increased amount, and as much as 120 ounces may be passed in the twenty-four hours; oftener it amounts to some 80 ounces. The urine is clear, pale, and of low specific gravity, usually about 1010, but it may vary a few degrees on both sides of this. Micturition is more frequent than normal, and is sometimes the symptom which draws the attention of the patient to the underlying ailment. There may be increased frequency of micturition by night, but that is not so frequent a symptom in this affection as it is in an enlarged prostate and in other diseases. The urine usually contains albumin in small amount, and it is not uncommon for a mere trace to be present. More usually larger quantities than this are present, and quite sufficient in amount to be recognised by any ordinary test. Larger quantities of albumin, such as a third or a quarter, are sometimes seen, but this increased amount is often really associated with an attack of subacute nephritis complicating the chronic and underlying malady. In the experience of the writer the amount of albumin is never so large as that seen in the second variety of chronic Bright's disease, although the other characters of the urine are very similar in the two classes of cases. Albumosuria is not uncommon in granular kidney, and in quite exceptional instances the amount af albumose excreted may be large. Casts in the form of granular and hyaline casts are

usually present. In rare cases the urine may contain no albumin, and this is not uncommon as a temporary phenomenon, so that in order to exclude granular kidney it is essential that more than one examination of the urine should be made. Quite exceptionally cases are sometimes seen where death has resulted from this malady, and yet where the urine has been free from albumin during the last illness.

Hæmaturia of varying degrees of severity is not uncommon in granular kidney, and occasionally large quantities of blood have been observed. In some instances the occurrence of this profuse hæmaturia associated with general wasting has led to the erroneous diagnosis of malignant disease of the kidney. The hæmorrhage may not only be profuse in amount, but may be also quite sudden in its occurrence, and it has been supposed that the blood is sometimes derived rather from the pelvis of the kidney than from the kidney substance.

Hæmorrhage from other mucous membranes, especially from the nose, is not uncommon in granular kidney, and it has been stated that occasionally hæmoptysis may occur. The occurrence of a profuse hæmorrhage will often relieve not only the headache and cardiac distress often seen in these patients, but it may even lead to an amelioration in any uræmic symptoms that may happen to be present.

Ocular symptoms.—Neuroretinitis and retinal hæmorrhages are common in cases of granular kidney, and the first symptom for which the patient may seek advice may be for loss or diminution of the acuity of vision from the occurrence of such retinal changes.

The **diagnosis** of granular kidney is usually arrived at from the association of the physical signs of cardiac hypertrophy and high-tension pulse with the presence of a dilute urine containing small quantities of albumin. Mistakes are liable to occur owing to the urine not being examined sufficiently carefully in patients with symptoms of impairment of the general health only.

In cases presenting cardiac symptoms it is often difficult to determine whether the cardiac or the renal disease is the primary malady, and a case of granular kidney with failing heart may be confounded with a case of mitral disease with passively congested kidneys. In many cases the presence of some evidence of increased tension, and also the changes in the vessels of the fundus oculi, may reveal the presence of granular kidney. Further, the urine in cases of passively congested kidney is usually dense and high coloured, that of granular kidney is of low specific gravity and pale, but these

differences are not invariably present, and even with the utmost care a mistake may be made.

The physical signs presented by cardiac hypertrophy in renal disease often resemble to a certain extent those seen in mitral stenosis, and it is possible that a condition of virtual mitral stenosis may exist in cases of cardiac hypertrophy secondary to granular kidney. Both in mitral stenosis and cardiac hypertrophy the first sound may be altered in character and become shortened, and, further, in the cardiac hypertrophy of renal disease the galloping rhythm is sometimes dependent on the presence of an added sound in the diastolic period resembling a presystolic murmur. This is another reason why primary renal disease may be confounded with primary heart disease.

The **prognosis** of granular kidney is most uncertain, owing to the impossibility of foreseeing the development of such complications as cerebral hæmorrhage, and even of acute uræmia, as this may develop with very great suddenness in patients who are apparently well. On the other hand, patients with granular kidney may live for many years, not only in moderate comfort, but even in fair health. Unfavourable signs are the presence of well-marked cardiac hypertrophy, and especially of cardiac dilatation. Further, the presence of marked albuminuric retinitis is of unfavourable import, and in most of such cases the duration of life may be measured by months. The prognosis is very unfavourable where not only cardiac dilatation but dropsy also has supervened, although even in such cases temporary improvement and subsidence of the dropsy may occur as a result of treatment. Uræmia and secondary inflammations, pericarditis, peritonitis, etc. in granular kidney are also most unfavourable in their issue.

One of the first indications of **treatment** of granular kidney is the avoidance of strains of all kinds. Cerebral hæmorrhage may occur as a result of strain experienced during exercise or even as a sequel of straining at stool. Many patients suffering from granular kidney in its earlier stages are still very vigorous, and tend to lead a more active life than is good for them, and it is essential in the treatment of this disease that such patients should not indulge in fatiguing pursuits, mental or physical. The diet should be of the simplest character, all highly seasoned articles of food must be avoided, and meat should be taken in small amount, although in an ordinary case it is not necessary to cut off meat from the diet unless complications such as cardiac dropsy or uræmia are present. In all such instances a milk diet is indicated, and should be persisted

in until the complete subsidence of all such unfavourable symptoms. Milk, milk puddings, fish, chicken, should form the staple articles of diet in a person suffering from granular kidney, even if his health be apparently good. Alcohol should be avoided absolutely, and tea, coffee, and cocoa should only be taken in small amount, but the patient should be encouraged to drink freely of simple diluents and mineral waters. It is most important to promote free action of the bowels in these cases in order to relieve the vascular high tension, and for this purpose saline purgatives in the form of sodium phosphate, sodium sulphate, or seidlitz powders are most useful.

The high tension may be also relieved by the administration of nitrites, especially nitro-glycerine and nitrite of amyl, erythol tetranitrate is also useful. Aconite and iodide of potassium may be used for this purpose, but are not of the same value.

The treatment of cases suffering from cardiac failure is difficult, but in the vast majority of cases the same remedies may be used as in other forms of cardiac failure, especially digitalis and strychnine. On theoretical grounds there are objections to the use of digitalis, but in practice beneficial results are often obtained. Strychnine is also of considerable value in these cases.

The uræmia arising in granular kidney may be treated on the same lines as when complicating other renal diseases, but venesection is especially useful in the uræmia developed in this affection.

CYSTIC DISEASE OF THE KIDNEY

The **symptoms** in cystic degeneration of the kidneys present a great deal of variety. In some the development of the cysts and the increase in the size of the kidney thereby produced, lead to the formation of such a large mass that the patient seeks advice either on account of the mere size of the tumour or on account of the weight and discomfort produced by its presence. In other instances symptoms are produced by the pressure of the enlarged kidney on surrounding structures, especially the colon, and thus intestinal obstruction may be produced. The most common group of symptoms consists of pain in the back, impairment in the general health, such as loss of strength, wasting, together with the occurrence of hæmaturia. The hæmaturia may be not only profuse in amount, but may also be the first symptom to attract the patient's notice to the underlying disease. It is by no means present, however, in all cases. Nausea and vomiting often become prominent symptoms

towards the end of the malady, and it is usual then for other uræmic symptoms to occur, although these may vary much in type. In some delirium, convulsions, and coma are seen, while in others the clinical picture resembles more that seen in calculous obstruction, *i.e.* so-called latent uræmia. Dropsy is usually absent, but slight subcutaneous œdema may be present. The urine is usually abundant and of low specific gravity, containing a trace of albumin, and resembling in its general characters that secreted in granular kidney. In the terminal stages of the disease, when uræmia is present, the quantity of urine secreted may undergo great diminution, and thus partial or complete suppression may occur. It is not unusual for cystic kidneys to produce no symptoms at all for many years, and then for uræmia to develop suddenly. In others the malady never produces any symptoms, and the condition is discovered accidentally in the post-mortem room.

The **diagnosis** of cystic kidneys is usually arrived at from the detection of more or less symmetrical tumours occupying the loins, together with the changes in the urine described above.

The tumours are usually soft, and frequently the irregularities on the surface caused by the cysts can be detected, although fluctuation is not necessarily or usually present. Greater difficulties are experienced when the disease is either more advanced on one side than the other, or in rare instances where it reaches a high degree of development on one side, the opposite kidney being scarcely affected at all. In such cases the diagnosis can only be a matter of conjecture, as it would be almost impossible to differentiate the condition from a hydronephrosis or other renal tumour.

The **prognosis** depends essentially on whether the malady is causing renal symptoms or not at the time when the patient comes under observation. If uræmia be present, even in its mildest forms, the duration of life will probably be very short. If, on the other hand, the only symptoms present are pain in the back and occasional hæmaturia, life may be maintained not only for months, but sometimes for years. In all instances the prognosis is very uncertain, owing to the possibility of the sudden onset of uræmia.

There is no **treatment** for cystic kidneys beyond care in dieting on the general principles followed in the treatment of Bright's disease. In the very rare instances where the disease has been found to be unilateral the affected kidney has been sometimes successfully removed. This treatment, however, is hardly possible unless an exploratory operation has been undertaken in order to determine the nature of a unilateral renal tumour.

RENAL GROWTHS

The essential **symptoms** presented by malignant disease of the kidney are pain and hæmaturia, associated with the presence of a renal tumour. They are, however, not necessarily present throughout the illness, and hæmaturia may be absent during the whole course of the disease in as many as half the cases. The *pain* is especially felt in the loin, and not uncommonly the patient seeks advice on this account. Attacks resembling those of renal colic are not uncommon, and are usually dependent either on the passage of fragments of growth or else of blood clot. The *tumour* produced by malignant disease of the kidney has the usual characters of a renal tumour, and it is often possible to make out the position of the colon crossing the tumour both by palpation and by percussion. A tumour of variable size can usually be detected at the time when the patient first comes under observation. Varicocele on the same side as the renal tumour is a common phenomenon dependent on interference with the return of blood through the spermatic vein caused by the tumour. *Hæmaturia*, although frequently present, is not invariable, and it may be quite scanty in amount. On the other hand it may be persistent, occurring daily for months, or in some instances years. The most usual condition, however, is for attacks of hæmaturia lasting for several days to occur from time to time. *Thrombosis of the renal vein* occurs not uncommonly in malignant disease of the kidney, and in some instances the clot may spread into the vena cava, leading to partial or complete thrombosis of this vessel, and so causing distension of the veins of the abdominal wall and œdema of the legs.

The duration of malignant disease of the kidney is usually less than a year after symptoms have declared themselves, but it is not very exceptional for cases even of carcinoma to live for many years after the first onset of hæmaturia.

Course.—Death usually occurs as a result of cachexia and asthenia, but in some instances intestinal complications, such as obstruction, may arise, or death may result from the effects produced by secondary growths in various parts of the body.

The **diagnosis** is usually made on the co-existence of the three symptoms of pain, hæmaturia, and the presence of the tumour, but even under these circumstances mistakes may arise and the disease be confounded with hydronephrosis associated with calculi, since profuse hæmorrhage into the hydronephritic sac may sometimes

occur, and so produce a large and apparently solid renal tumour. Where a tumour is not detected the disease may be confounded with calculous and other renal affections. If a renal tumour be present and hæmaturia absent, a differential diagnosis from other abdominal tumours based on the physical signs of renal tumours has to be made.

The **prognosis** depends not only on the time when the disease is first recognised, but also on the variety of the malignant disease present. There are many instances on record of the successful removal of a malignant growth that has begun in an adrenal rest, and the outlook after nephrectomy is better in these cases than in those of the other forms of sarcoma and carcinoma of the kidney. Further, the operation of nephrectomy is more successful in cases of sarcoma than in those of carcinoma.

The **treatment** is essentially surgical, involving the removal of the affected kidney if the disease be recognised sufficiently early. In some instances, and more especially in cases of adenomatous growths beginning in an adrenal rest, partial nephrectomy has been successful, since such tumours can sometimes be shelled out from the remaining renal substance.

BENIGN TUMOURS of the kidney usually give rise to no symptoms. In very exceptional instances adenomata are seen which have undergone cystic degeneration, and may produce clinical symptoms somewhat resembling those seen in cystic disease of the kidney.

TUBERCULOUS DISEASE OF THE KIDNEY

Symptoms.—Tuberculous disease of the kidney often only produces symptoms of pyelitis, but not infrequently in addition to these a renal tumour is formed, either owing to the development of large masses of caseous tubercle in the kidney itself, or as a result of the blocking of the ureter by tuberculous masses and the distension of the organ with urine. Such a hydronephrosis may also become secondarily infected and a pyonephrosis develop.

One of the most characteristic features of tuberculous pyelitis is great *frequency of micturition;* this, no doubt, in some instances is dependent on co-existing tuberculous disease in the lower urinary tract. *Pain* in varying degree is usually present, and is often constant in the loin, but in addition to this attacks resembling those of renal colic often occur, dependent on the passage of portions of caseous tuberculous material down the ureter. Constant lumbar

pain is perhaps more marked than in most instances of calculous disease, and the intermittent renal colic-like attacks are less marked and less severe than those seen in true renal colic dependent on stone. *Pyuria* is always present in variable amount, and by proper methods of staining or by inoculation the presence of tubercle bacilli can often be demonstrated, and this is one of the most valuable means we possess for determining with certainty the existence of renal tuberculosis. *Hæmaturia* is usually present during some period of the course of the illness, but it is often slight in amount, the blood not being so abundant as the pus; in exceptional instances profuse hæmaturia may occur and may be the first symptom to attract notice.

The *general symptoms* of a tuberculous affection are especially marked when the malady is fully established. Great emaciation occurs together with considerable fever and much sweating. Involvement of the lungs, of the bowel, and also of the spine, occurs as a secondary result.

In **diagnosis** the main difficulty is to distinguish between tuberculous and calculous pyelitis. In the former pus is usually abundant and blood scanty, and the duration of calculous pyelitis is often very much more prolonged than that of tuberculous disease, since in this malady generalised infection is liable to develop. In many cases the diagnosis can only be made with certainty by the detection of tubercle bacilli in the urine. The most reliable method is that of inoculation of the subjected urine into animals, *e.g.* the guinea-pig. If a pyonephrosis have developed the absence of pus from the urine may render the diagnosis from other renal tumours difficult. It is not uncommon in pyonephrosis, whether of tuberculous or other origin, for the urine to be free from pus owing to the complete blocking of the ureter. Examination of the lower genito-urinary tract and the detection of tubercle in the epididymis in the male, or of a thickened ureter in the female, will often enable a correct diagnosis as to the nature of a renal tumour to be made.

The **prognosis** is very grave unless the case is seen quite at the outset and when the disease is limited to one side. It is very commonly bilateral at the time when the patient seeks advice. The duration of life is often only measured by months from the time when the disease is first recognised; patients, however, may survive for many years.

Treatment.—Removal of the affected kidney, where the disease is unilateral, offers the best chance of successful treatment, but it is extremely difficult to determine with any degree of certainty whether the disease is bilateral or not. Occasionally catheterisation of

the ureters has been successfully used to determine this point. The development of pyonephrosis in the course of tuberculous disease of the kidney will also need surgical interference. Medically the disease can only be treated on the general principles underlying the treatment of tuberculous disease in other organs, together with measures directed to dilute the urine and to render it as little irritating as possible. For this purpose alkalies and such drugs as urotropin are useful.

PYELITIS AND PYONEPHROSIS

Symptoms.—In uncomplicated pyelitis fever, pain in the back and frequency of micturition are the chief symptoms. The *pyrexia* is usually moderate, reaching 101° to 102°, but at the onset the fever may reach a height of 104°. The *pain* is usually lumbar and is commonly bilateral, although at first it may be limited to one side. *Tenderness*, especially in the lumbar region, is present and is greatly aggravated by pressure. Some tenderness may also be elicited on deep abdominal palpation in the region of the kidney. *Micturition is frequent* even when there is no evidence of the involvement of the lower urinary tract and bladder. Pus is present in the *urine* in variable amount and the quantity is often large. The urine is usually acid and blood may be absent.

Although simple idiopathic pyelitis sometimes occurs it is more often seen as a complication of other conditions, such as the presence of stone or of tubercle in the kidney. In these circumstances the symptoms may be considerably modified by the underlying disease, and hæmaturia is more liable to be present.

The **duration** of simple uncomplicated pyelitis is very indefinite, but the condition usually lasts for some weeks, and the temperature chart may have a very irregular course, febrile paroxysms occurring at irregular intervals.

Complications.—Pyelitis may become complicated by the development of pyonephrosis, although in some cases of pyonephrosis the condition develops without any initial illness characterised by the presence of pus in the urine, in other words, the inflammatory swelling produced by the pyelitis apparently blocks the ureter from the commencement. Certainly in most cases of acute pyonephrosis developing suddenly in persons previously healthy and unaccompanied by any other renal disease, it is rare to find pus in the urine. Pyelo-nephritis may ensue as a complication of pyelitis, especially

when the latter is dependent on an ascending infection from the lower urinary tract. Abscess of the kidney may also occur. In both these conditions the general symptoms of illness are far more marked than in cases of simple uncomplicated pyelitis. Wasting, rigors, and a general cachectic appearance are all present. Abscesses of the kidney and pyonephrosis may lead to further complications, such as retroperitoneal or even subphrenic abscess. When the condition is not recognised and promptly treated pyæmia may develop.

Diagnosis.—The presence of pus in acid urine accompanied by tenderness in the loin and frequency of micturition are the usual signs upon which the diagnosis of pyelitis is made. The urine may be alkaline where pyelitis develops as a complication of cystitis, and in some instances of uncomplicated cystitis the urine may remain acid.

The diagnosis of pyonephrosis and of abscess of the kidney is usually based on the presence of a renal tumour associated with the general signs of suppuration. Pus may be completely absent from the urine, however, in both of these conditions.

Prognosis.—The prognosis depends mainly on its cause and also on the question whether any complication such as pyelo-nephritis be present. Cases of acute pyelitis arising independently of the presence of stone, and not associated with an acute specific, are sometimes seen in otherwise healthy persons. Such cases, even if accompanied by high fever, usually run a benign course and subside in the course of a few weeks. Pyelitis depending on an ascending infection of the urinary tract is more serious than that associated with the presence of gravel or stone; the latter may exist for many years without causing any very serious consequences.

Treatment.—Many cases of pyelitis require surgical treatment owing to their cause, *e.g.* the presence of calculi, or else owing to the presence of complications, *e.g.* pyonephrosis. The main principles of medical treatment are to render the urine as dilute and unirritating as possible with barley water, together with infusion of buchu and citrate of potash in drachm doses. Urinary disinfectants, more especially urotropin, are often of great service. The salts of benzoic acid may be used for the same purpose. Stimulants are not to be taken if the disease be acute, and a simple milk diet should be ordered. In the more chronic forms of the malady a light diet, consisting mainly of fish, milk, and farinaceous foods, should be taken, meat being reduced to a minimum.

NEPHROLITHIASIS

Symptoms.—Renal calculi may exist without the production of any symptoms or any ill effects, and this is more especially the case where the stones are of large size. Further, it is not very unusual for serious and permanent effects to be produced on the kidney by the presence of calculi without the development of any symptoms. Thus in bilateral calculous disease it is not uncommon for the kidney on one side to be shrunken and atrophied, with the pelvis full of stones, and yet the patient may never have experienced symptoms of renal colic on that side. In many instances of complete anuria dependent on the presence of calculi, the patient only gives a history of renal colic on the one side, and makes no allusion to having experienced similar discomfort at an earlier period on the opposite side. Complete anuria may be dependent on simultaneous blocking of both ureters by calculi, or on the blocking of the ureter of the only existing kidney, its fellow being absent; but in a very considerable number of cases calculous anuria is dependent on the blocking of the ureter of the only efficient kidney, calculous disease having led to the destruction of its fellow without the production of any symptoms.

Although large stones may be present without the production of any symptoms, this latency is not limited to these, and in some instances quantities of small stones may be passed with little or no discomfort. It is more usual both for the presence and passage of stones to be accompanied by the development of symptoms. *Pain*, especially across the loins, or limited to one loin, is the most constant symptom. It may amount to a little more than a continuous ache, or may be of such severity as to completely incapacitate the patient. The pain is usually not confined to the loin, but radiates in a zone-like manner round the body in a slightly oblique direction from the loin towards the umbilicus. This pain may be accompanied by cutaneous tenderness which affects a band-like area, bounded above by a line from the twelfth dorsal spine to the umbilicus, and below by a line extending from the third lumbar spine to the symphysis pubis.

This variety of pain and tenderness is usually produced by the presence of stones in the renal pelvis. If the stone become engaged in the ureter a second variety of pain is present, which extends downwards into the scrotum and groin, and in exceptional instances may radiate down the back or outer surface of the thigh. Instances have indeed been mentioned in which the pain of renal

colic extended as far as the heel, but these are very exceptional. The pain of *renal colic* is usually paroxysmal, but it is not uncommon for patients suffering from this disease to have a constant, dull, aching pain and soreness in the loin, with exacerbations of acute radiating pain from time to time. The paroxysm of pain, which may be of intense severity, is accompanied by rigidity of the abdominal muscles, *nausea*, *faintness*, *and copious perspiration*, and it is not very unusual for it to be accompanied by a *rigor*, in which the temperature may rise to as high as 103°. The nausea and faintness may be very severe, and the patient during the severe paroxysm may become quite collapsed. *Vomiting* is often a very marked symptom. During the height of the pain there is great desire to pass water at very short intervals, and a considerable amount of *tenesmus* may be present. The flow of *urine* is greatly diminished, and what is passed often contains blood, generally in small amount, though profuse hæmorrhage may occur. As mentioned above, complete suppression of urine may be produced during an attack of renal colic, and in some exceptional instances the occurrence of complete suppression is the first symptom of ill-health, the patient never having experienced an attack of renal colic before. When complete suppression occurs associated with the presence of calculi the patient does not present the ordinary symptoms of acute uræmia, but notwithstanding complete cessation of the urinary flow he remains conscious, and no delirium, coma, or convulsions ensue. Life may be prolonged for as long as a week or even ten days, notwithstanding that during the whole of this time no urine may be secreted. The most marked effects produced are a fall in the body temperature, a sensation of great lassitude and weakness, the development of extreme myosis, and occasionally the occurrence of slight œdema. Death may occur quite suddenly from respiratory failure without the prior development of any other symptoms. Some nausea, twitching, and dyspnœa may occur just before the end.

The impaction of a renal calculus may be followed, not by the development of calculous anuria, but by that of hydronephrosis.

Diagnosis.—Renal colic has to be distinguished from other forms of abdominal colic, and this may usually be done not only from a consideration of the distribution of the pain, but also by the occurrence of hæmaturia. The detection of tenderness over one kidney is often a valuable indication, although it is said that the symptoms may occur on the opposite side to that on which the stone is impacted. Attacks of pain occur in movable kidney resembling those seen in calculus, and rare instances are known in which attacks

of pain, apparently indistinguishable from those of renal calculus, have occurred where no morbid lesion could be discovered in the kidney. It is thought by some that the hard, irregular oxalate of lime calculus is liable to produce more severe symptoms than those associated with the uric acid stone.

The **prognosis** of calculous disease of the kidney depends, to a great extent, on the presence or absence of complications such as pyonephrosis, pyelitis, etc. There is necessarily considerable uncertainty in the prognosis owing to the possible development of calculous anuria, and the conditions that determine the occurrence of this are not known. Provided the kidneys are healthy, as shown by the state of the urine, the prognosis of calculous disease is, on the whole, good as to life, and suitable treatment may lead to the cure of the malady.

Treatment.—In cases of pain in the back, dependent on the presence of minute calculi, great relief may be experienced by the administration of citrate of potash and free dilution of the urine, together with the avoidance of a large meat diet. If the irritation be produced by the presence of oxalate of lime crystals, the treatment must be directed to prevent the patient taking articles of diet rich in oxalates, and at the same time treatment directed to diminish or prevent gastric fermentation should be employed.

Medicinal treatment is probably useless for the purpose of procuring the solution of actual stones, although such measures as the continuous administration of alkalies may be of great value in cases of gravel, in order to prevent their formation.

Surgical measures must be employed for the treatment of calculi giving rise to repeated attacks of renal colic and there is evidence that the patient is not passing the stones.

During the actual attack of colic hot applications to the loin and hot baths may be useful to relieve the pain if this be slight in amount, but in most cases either morphia or chloroform will be called for.

HYDRONEPHROSIS

Symptoms.—The hydronephrosis seen as a complication of uterine carcinoma or fibroids, or of ovarian disease, is very often unaccompanied by symptoms, owing to the very gradual manner in which the pressure effects are produced. Not only may all symptoms be absent, but the composition of the urine may undergo little or no change if the hydronephrosis be unilateral. When

bilateral and partial the specific gravity of the urine falls. In the more severe forms of hydronephrosis, where the obstruction to the ureter is complete, as in calculous obstruction or in some forms of movable kidney, the symptoms produced are much more severe. Pain is a marked feature, especially in the back and in the hypochondrium, but it does not usually radiate down into the groin or testicle. Nausea and vomiting are also present, and these three symptoms may be so severe in the earlier stages as to suggest the occurrence of intestinal obstruction. When the hydronephrotic kidney is of large size pressure may be exercised on the surrounding intestines and constipation result. In unilateral complete hydronephrosis it is not usual for the quantity of the urine secreted to undergo much change, the remaining healthy kidney secreting twice its normal amount.

Hydronephrosis may be intermittent, the obstruction may give way, and the tumour subside with the passage of a very large quantity of urine. This is more especially seen in the hydronephrosis accompanying calculous obstruction and that met with in movable kidney.

In double partial hydronephrosis, as for instance in that complicating pelvic disease, uræmia is apt to ensue. The symptoms generally are of a subacute type—slight delirium, muttering, twitching, nausea, and finally drowsiness, ending in death from coma.

The **diagnosis** of hydronephrosis turns largely on the recognition of the fact that the tumour is renal. A hydronephrotic kidney usually fills up the loin to a marked extent, and fluctuation can often be detected. In such cases the main difficulty is to distinguish between a hydronephrosis and a perinephritic collection.

The **prognosis** as to life depends essentially on the cause, and more especially on whether the disease is unilateral or not. Where the condition supervenes as a result of carcinoma of the uterus or other pelvic growth, the prognosis is necessarily most serious. On the other hand, when seen as a complication of calculus or movable kidney the outlook is favourable. Where the kidney has undergone great dilatation as a result of hydronephrosis, although it may not lead to death, the kidney is more or less permanently damaged, and often its value as an excreting organ is very seriously impaired.

The **treatment** of unilateral hydronephrosis consists essentially in draining the distended organ, or in the performance of nephrectomy. The former is the more usual procedure, but

sometimes the impacted stone cannot be dislodged, and it may be necessary to remove the distended and sac-like organ.

Bilateral hydronephrosis, the sequel of pelvic disease, is not amenable to treatment except in so far as relates to the pelvic disease causing it.

JOHN ROSE BRADFORD.

DISEASES OF DUCTLESS GLANDS

In an earlier portion of this work (Vol. II.) certain disorders of nutrition and of the blood have been described which are associated with morbid conditions of "ductless glands." It may be recalled that the well-defined clinical states of Graves' disease (*loc. cit.* p. 155), myxœdema (p. 150), Addison's disease (p. 161), and acromegaly (p. 265), have been more or less satisfactorily demonstrated to be dependent on changes in the thyroid, suprarenals, and pituitary body respectively.

Although the most important diseases in which ductless glands are involved have been already dealt with, there still remain many localised morbid conditions to be considered. The thymus is an organ which, whilst epiblastic in origin, early becomes infiltrated with lymphoid cells, and is probably, during its comparatively transient existence, more concerned with hæmopoiesis than with metabolism. Indeed, in its developed form scattered epithelial relics in the midst of the predominant lymphoid tissue alone recall its kinship to such an organ as the thyroid, and it therefore remains uncertain whether, like that gland, it does produce any internal secretion. But the spleen, lymphatic glands, and thymus are only parts of the vast lymphatic system that pervades the whole body, and includes the extensive stratum of lymphoid tissue underlying mucous membranes, as well as the larger localised collections of such tissue that compose the tonsils and the solitary and agminate glands of the intestines. No doubt, too, the bone-marrow should also be regarded as belonging to the same class.

LYMPHATISM

The condition described as *lymphatism*, or the *status lymphaticus*, is one in which there would appear to be a general hyperplasia of the lymphatic textures. The subjects of it are children, anæmic and delicate, perhaps unhealthily inclined to obesity. They would seem to possess but little vital resistance, and to be liable to sudden death from comparatively slight causes. At any rate it is to the existence of this "constitutional" vulnerability that have been referred cases of death during the administration of anæsthetics, or from the shock of operations of a trifling character—cases in which

the only morbid appearances are confined to the lymphatic system. The thymus gland (see p. 669) is very frequently enlarged, and the cases of sudden death from heart failure where little or nothing abnormal has been found post-mortem beyond thymus hyperplasia may, according to Paltauf and others, be really due to lymphatism. This is, moreover, often found in fatal cases of diphtheria and laryngismus stridulus, to which diseases it may be said to impart an unfavourable aspect. Of the latter, indeed, it is held by many to be an important predisposing cause. The internal lymphatic glands, such as the bronchial and mesenteric, the tonsils, post-pharyngeal and intestinal lymph-glands, are notably enlarged. There is a like hyperplasia of the spleen, of which the Malpighian corpuscles are markedly prominent. The thymus is soft and has a whitish appearance on section, whilst the "yellow" marrow normally met with in the bones of young subjects is here replaced by "red" marrow. The condition may be associated with rickets and also with enlargement of the thyroid gland, and hypoplasia of the heart and aorta (Osler).

DISEASES OF THE SPLEEN

Morbid Anatomy and Etiology

Variations in shape and size.—The spleen is an organ which may exhibit many departures from what may be considered its normal size and shape. In respect to *size* there are rather wide limits consistent with health; indeed, its structure and vascular relations permit of its becoming more or less filled with blood in harmony with a relative state of turgescence or otherwise of the abdominal vessels. It is recognised, for instance, that, during digestion, the spleen becomes more turgid, acting as a sort of reservoir to the blood that flows in increased volume to the stomach; and the nature of the investing capsule of the organ permits this distensibility. In *shape* it may be more flattened and elongated than usual, reverting somewhat to the form of the organ obtaining in lower animals; whilst great variations occur in the depth of the characteristic notch or notches to be found on its anterior margin. Lastly, as regards *site*, the organ may be congenitally displaced, as in cases of transposition of viscera, or may occupy a lower position than natural, being then more completely invested by peritoneum. Cases of so-called "wandering spleen"

are often of this nature, the organ coming to lodge as low as the pelvic brim, and being detectable by vaginal examination. This condition occurs mainly in women, and is due to abnormal length, or looseness, or accidental tearing of the attachments of the organ. Pregnancy, as in the case of displacements of the kidney, is believed to be a frequent cause. The spleen is seldom enlarged in this condition. In extremely painful cases the organ has been excised. Splenopexy should, however, be tried first.

In connection with such anatomical peculiarities mention may be made of the existence of small rounded masses of splenic substance which are by no means infrequent. They occur in the peritoneal folds in the vicinity of the spleen, and are known as "splenculi," their structure reproducing the characters of the main organ. It seems probable that the ill effects of splenectomy are compensated for by the existence of such accessory spleens taking on the functions of the extirpated organ.

Acute congestion and inflammation. — These terms as applied to the spleen must be used with reservation. The organ does, undoubtedly, undergo rapid vascular engorgement, both physiologically during digestion, and in certain diseases where such "congestion" is associated with parenchymatous changes, which, in other organs, would be styled "inflammatory." But it is open to question whether a less specific term, such as "acute swelling" or "softening," would not be more suitably applied to the hyperæmic conditions that mainly arise in consequence of infective febrile diseases. In such diseases enlargement of the spleen with a greater or less diminution of its normal consistence has been long known to occur. It was held to be characteristic of the state of high fever, and was even attributed to the effect of the heightened temperature of the body. The true explanation of the proneness of the spleen to be so altered in "fevers" lies, however, in the fact that, owing perhaps to its peculiar vascular mechanism, it affords a resting-place, or perhaps, more strictly speaking, a filter for the specific microbes, and is thus directly subjected to the action of their toxins. The most striking example of this is to be seen in bovine anthrax, the synonym of which, "splenic fever," denotes the most characteristic fact in its morbid anatomy. Here the enormously enlarged organ has its blood-vessels filled with the bacillus anthracis, which grows in the channels and sinuses with marvellous rapidity. And in most bacterial infections it is possible to obtain the organisms from the splenic blood when it may be difficult to get them from the general circulation. Hence puncture of the

enlarged spleen with a hollow trocar and bacteriological examination of the material removed have been much employed on the Continent in the diagnosis of typhoid fever. To this end it is even more accurate than the agglutination reaction, though, for obvious reasons, of much less practical utility.

The anatomical changes undergone by the spleen in consequence of bacterial invasion are very similar in the various infective diseases, although in some they are carried to a far greater extent than in others. In the first place there is marked hyperæmia; secondly, a proliferation of the cells of the splenic pulp and Malpighian corpuscles, followed, thirdly, by necrotic and degenerative changes in these cells. To the naked eye the organ is more or less enlarged, full of blood, sometimes very dark in appearance, but more often of a reddish-pink and greatly softened, so that it is almost, if not quite diffluent when incised; and the softened pulp may be washed out of the trabecular framework by a stream of water. The organ is so soft that it is often torn in the attempt to remove it, which may have given rise to the belief that it occasionally ruptures spontaneously from over-distension of the capsule by the swollen pulp.

The cell proliferation, which is probably excited by the presence of bacteria and their toxins, is mainly of the lymphoid elements, and the phagocytes thus produced may in their turn serve to repel the invaders, and help to free the body from them. Whether the spleen takes any further part, and if so, to what extent, in the production of immunity in infective diseases is a subject which has been much debated.

Amongst the diseases in which this form of splenic enlargement and softening is most prone to occur may be mentioned relapsing fever, typhus fever, enteric fever, pneumonia, septicæmia, and malignant fevers (variola, scarlet fever, measles), diphtheria, and ulcerative endocarditis.

In malarial fevers the implication of the spleen, which forms so characteristic a feature, is doubtless allied with the fact of the organism of these diseases being a hæmatozoal parasite. The splenic enlargement in typhoid fever does not run *pari passu* with the course of the disease, but reaches its maximum in an early stage, being usually distinguishable during the first week. Its extent bears no relation to that of the intestinal lesions.

In relapsing fever the spleen is particularly large and contains the pathogenic spirillum. It was swollen in 96 per cent of cases in the great epidemic in Breslau (Litten).

When an infective disease causes acute enlargement of the spleen in a patient who is already the subject of aortic regurgitation, pulsation of the organ has upon several occasions been noted.

Chronic congestion and induration. — Long-continued venous congestion produces in the spleen similar structural changes to that which it entails in other viscera, *e.g.* the liver and kidneys, —namely, an induration of the organ from an increase of its connective tissue. This is usually combined with some thickening of the capsule, and should this occur to any extent the size of the spleen may not be much altered. Often, however, there is notable enlargement, the organ retaining its shape. It is dark in colour and very tough in consistence. Microscopically the trabeculæ throughout the organ are seen to be greatly thickened, and the pulp-tissue is proportionally diminished, whilst the venous sinuses are dilated and filled with blood. The condition is perhaps best marked in cases of mitral disease, and in those of dilatation and hypertrophy of the right ventricle from obstruction to the pulmonary circulation, as in emphysema. Indeed, it is safe to say that wherever there is a nutmeg liver there is also an indurated spleen, and it is probable that the latter follows upon the portal obstruction due to the impeded hepatic circulation. It is to be noted, however, that in most cases of compensated valvular heart disease the spleen is smaller than normal, unless infarctions are present.

The interference with the portal circulation produced by cirrhosis of the liver has the same result, and in some cases of cirrhosis the spleen may attain considerable dimensions. It has been pointed out that portal obstruction alone can hardly account for the change undergone by the spleen, since this organ is often more enlarged in the earlier than in the later stages of hepatic cirrhosis. It is suggested, therefore, that the toxæmic state that induces the cirrhotic change in the liver may also operate in producing some of the induration of the spleen, the condition being regarded as one of chronic inflammation, similar to that observed in chronic infectious disorders, as syphilis and malaria. In support of this view is the microscopical evidence of hyperplasia of the cellular elements as well as of the interstitial tissue in the greatly enlarged organ, whereas in the purely congestive indurated spleen the splenic pulp is, as stated, atrophied.

On the other hand, some of the largest spleens are those met with in conditions of portal obstruction where toxæmic influences can less plausibly be invoked, *e.g.* in thrombosis of the portal vein.

The *malarial spleen* is not only uniformly enlarged and indurated, but is also deeply pigmented, the pigment being due to the disintegration of the red blood corpuscles that occurs in malaria. It may attain considerable dimensions; there is always much perisplenitis.

It is noteworthy that the characters of the spleen in so-called "splenic anæmia" much resemble those due to chronic toxæmias, namely, an indurative interstitial inflammation. Some of these cases end up with all the characteristic signs and symptoms of cirrhosis of the liver. It is with this type of disease that the name of Barti has been associated. The change which occurs in syphilis and causes the enlargement of the organ, especially in congenital syphilis, is also interstitial; it is often associated with lardaceous degeneration. Lastly, in rickets the enlarged and firm spleen, to which Sir W. Jenner gave the name "albuminoid," is structurally indistinguishable from these indurative interstitial inflammatory lesions that are apparently evoked by toxæmic agencies.

Infarctions.—Owing to the arteries of the spleen being of the nature of end-arteries, *i.e.* each branch supplying a definite area of its own, the results of blocking of these vessels by embolic or thrombotic plugs is to produce an *infarct;* and the condition is one of the most common lesions to which the spleen is liable. The large size of the splenic artery and its proximity to the heart may explain the frequency with which emboli coming from the valves or chambers of that organ find their way into this vessel. It may safely be said that there are few cases of fatal mitral stenosis in which splenic infarcts of various sizes and ages are not to be found. In size the infarct may range from a small wedge-shaped mass of the dimensions of a cherry-stone to one which involves the whole thickness of the organ. The base of the wedge is directed towards the periphery, from which it may be separated by a narrow layer of splenic tissue immediately underlying the capsule. In its early stages the infarct may have a pale yellowish appearance surrounded by a deep red zone, and be found to consist of granular detritus of blood and cells held together by the trabecular strands which are swollen, and are, like the contained pulp, undergoing necrosis. In other cases the infarct is of a deep red damson cheese aspect, due to infiltration with blood. It is usual to speak of the former as the "anæmic" infarct, and the latter as the "hæmorrhagic," but, except for the amount of blood extravasation, there is no essential difference between them. Both are areas of necrosis corresponding to the arterial branch that is blocked. Where the infarct is

hæmorrhagic, Cohnheim believed that the blood gained entrance by a reflux from the veins in the immediate vicinity; but this view has been disputed. The infarct at its inception excites inflammatory reaction around it, so that frequently the capsule and the investing peritoneum opposite to it become inflamed, with consequent adhesions to the parietal peritoneum, whilst the splenic tissue surrounding the necrotic mass becomes condensed and toughened. As time goes by the caseating mass shrinks, until ultimately the only indication of the infarct may be a puckered and pigmented fibrous nodule near the surface of the spleen, which thus has a scarred appearance. It is not unusual to see the various stages of the process exhibited in the same spleen in several different foci.

If the source of the embolus and therefore the embolus itself be septic or infective, as in ulcerative endocarditis or pyæmia, then the infarct will suppurate. Most examples of *splenic abscess* have this origin; others are due to extension of the process from a neighbouring organ. Suppuration following general acute swelling or inflammation rarely occurs except in association with enteric or relapsing fever; although, as stated, the infections which cause acute swelling lead to necrotic changes in the cells. Diphtheria affords good examples of scattered focal necrosis of this kind.

Lardaceous degeneration. — The marked liability of the spleen to become affected by lardaceous or waxy degeneration (in 98 per cent of all cases, Litten) aids materially in the diagnosis of this condition during life, associated as it usually is with prolonged suppuration, chronic tuberculosis and syphilis, and less often with carcinoma and gastric ulcer. It mostly leads to enlargement of the organ, or at least to an increase in its weight, without, however, modifying its shape. In advanced cases it is so firm in consistency as to feel as solid as a kidney, its margins being rounded, and the organ when removed from the body retaining its shape, instead of being flattened. It thus retains the convexity of its outer and the concavity of its inner surface precisely as if it had been moulded in wax. There are two distinct forms of the affection depending on the structures which are chiefly the seat of the infiltration. In the one form—the *diffuse waxy spleen*—the cut surface presents an uniformly smooth and glistening, but still reddish aspect, and in consistency resembles uncooked bacon rind, a fact which led to the German term of "speckig" being applied to it. Here the change, which commences in the walls of the arteries and venous sinuses, ultimately involves

the pulp elements, so that in advanced stages the microscope only reveals masses of waxy material, giving the characteristic staining reactions with iodine and methylaniline, and wholly concealing the trabeculæ. The other form—the *sago waxy spleen*—is more common. It derives its name from the appearance presented by the cut surface of the organ, which from the numerous translucent spots with which it is studded looks as if grains of unboiled sago were embedded in it. This macroscopic appearance is due to the fact that here the waxy change mainly affects the lymph capillaries and the Malpighian bodies connected with them, these latter swelling and becoming throughout infiltrated with the lardaceous material. In the earlier stages the spleen may at first sight appear to be normal, but close inspection reveals the granules standing prominently out. On treating the section with a solution of iodine these spots assume a dark mahogany brown colour, contrasting with the paler tint of the unaffected surface. In advanced cases the masses are quite contiguous, but the change is never so uniformly spread as in the diffuse form.

Tuberculosis.—In acute miliary tuberculosis the spleen, without being notably, if at all, enlarged, may be stuffed with tubercles, which appear as minute semi-translucent granules, firmer and more resistant than the natural Malpighian corpuscles, for which they must not be mistaken. In such cases also small granules may be scattered over the splenic capsule. Less common are larger caseous masses which are in general more sparsely distributed, although occasionally these too may be very numerous. They are firm on section, opaque, and of a bright yellow colour. Miliary tubercle of the spleen attacks chiefly the Malpighian bodies, chronic the arterial sheaths.

Actinomycosis. — The characteristic nodules and abscesses produced by actinomyces have been met with in the spleen, but are very rare.

Syphilis.—Enlargement with induration from a general fibrotic change occurs both in inherited and acquired syphilis; and lardaceous degeneration is a concomitant of advanced stages of this infection. The spleen may also be the seat of gummata which have to be distinguished from caseous tubercle and infarcts. It is stated that the spleen is enlarged in 35 per cent of newborn babies suffering from congenital syphilis; there is at present much divergence of opinion as to the responsibility of this disease for the hypertrophied spleens so often met with in children during the first three years of life.

Leukæmia.—It may be recalled (see Vol. II. p. 325) that in this disease the spleen attains its greatest dimensions, being in a condition of hyperplasia in which the trabeculæ as well as the pulp share. The latter being engorged with leucocytes may give a stippled appearance to the cut surface, whilst the general consistency of the enlarged organ is firm. Areas of hæmorrhage and definite wedged-shaped infarcts are commonly met with in advanced cases.

Hodgkin's disease (Vol. II. p. 355). — The characteristic feature of the spleen in this disease, apart from its enlargement, is the presence within its substance of more or less closely aggregated masses of lymphoid tissue, the whitish colour of which is in marked contrast to the dark red tint of the splenic tissue.

Neoplasms.—Apart from capsular *fibromata* the spleen is very rarely the seat of primary growths. These fibromata consist of densely compacted layers of fibrous tissue, which may form opaque white smooth prominences on the surface of the organ, or may be so uniformly distributed as wholly to invest it within a dense sheath a quarter of an inch or more in thickness. The growth is as firm as cartilage, and has been likened to the lamellar structure of the cornea.

Secondary nodules of *sarcomata* and *carcinomata* may be met with, but it is remarkable how seldom the spleen is so affected as compared with the liver. Or the spleen may be invaded by cancerous growth proceeding from the contiguous stomach or suprarenal capsule.

Hydatid cysts are occasionally found, or their calcified relics. *Dermoid cysts* are rarely met with. *Serous* and *hæmorrhagic cysts* are also occasionally present, and when large have been mistaken for hydronephrosis.

Clinical Characters

There is not much to be said from the clinical and therapeutical standpoint regarding the various forms of splenic disease which have just been described. There are no characteristic symptoms directly referable to a disordered spleen, unless the blood states of leukæmia and of so-called splenic anæmia are to be so regarded. In all the other affections in which this organ is involved the symptoms are those of the disorder upon which the splenic affection secondarily depends. There may be a sense of weight or a dull aching sensation in the left hypochondrium when

the organ is much enlarged; or more acute pain accompanied, perchance, by a sudden rise of temperature may indicate the occurrence of splenic embolism. But apart from such inconstant indications the presence of disease of the spleen cannot be revealed without physical examination, which shows that the organ is abnormally enlarged. As most of its diseases lead to its enlargement the importance of such physical examination is obvious.

In this examination palpation and percussion are the methods employed. For palpation it is essential that the abdominal muscles be relaxed, to which end the patient should be recumbent, with the knees raised and flexed, and a pillow placed beneath the buttocks. Standing on the left side of the patient, the examiner places the right hand on the loin, and with the left on the anterior surface of the body beneath the costal arch gently depresses the fingers. Should the spleen be enlarged he will thus support it from behind and also determine the extent to which it projects below the ribs in front. In health the organ is not to be felt, but if it only projects a short distance below the ribs its smooth and rounded inferior border is readily detected.* Should the enlargement be considerable, then the anterior margin can be traced and the depression caused by the notch (or notches) distinctly felt. Some idea can also be gained of the firmness of the organ by the resistance it affords to pressure. In cases of acute swelling (*e.g.* in typhoid fever) there may be distinct tenderness experienced by the patient. It is also easy to ascertain whether the surface is smooth or irregular. Mostly, too, it is possible to move the organ forwards by pressure from behind; and, except in cases of great enlargement, or where adhesions have formed, it can be distinctly felt to descend with deep inspiration.

The solid tumours presenting in this region which are most likely to be mistaken for an enlarged spleen are tumours of the left kidney or suprarenal body. Neither is so mobile as the spleen, and in each case the anterior margin is more rounded and is not intersected by the notch, which, when well marked, is quite characteristic of a splenic swelling. Greater difficulties in diagnosis may, however, be caused by the presence of malignant or tuberculous masses in the omentum. These may exactly simulate an enlarged spleen both in size and shape, even the notches being accurately reproduced. In such cases a correct decision can only be arrived at, if at all, by a most careful study of the associated symptoms.

Percussion affords the means for further differentiation. Nor-

mally the area of splenic dulness is confined to the infra-axillary region over the tenth and eleventh ribs, and is traceable round to the back, the vertical depth of this band of dulness being about one and a half inch. When the spleen is enlarged this area is increased in all directions—upwards, downwards, and forwards—and is continuous with that obtained over the left hypochondrium. The area of dulness is never conterminous with the whole extent of the swelling as determined by palpation, owing to the contiguity of the stomach and intestines. It is in its relation to the latter that an enlarged spleen differs most markedly from a renal tumour. The spleen as it enlarges downwards is in contact with the abdominal wall lying in front of the intestines, whereas the kidney generally pushes the descending colon in front of it. The splenic dulness is therefore generally bounded in the flank by an area of resonance due to the colon; the renal dulness persists in the flank, whilst the anterior part of the tumour is overlaid by the resonant colon. It should, however, be borne in mind that sometimes the greatly enlarged spleen will displace the transverse colon in such a way that this comes to lie between the spleen and the abdominal wall. When much enlarged, as in leukæmia, the spleen may reach almost to the pelvic brim, and its anterior margin be felt beyond the linea alba; but even then there can generally be made out some resonance in the left flank, which is never present in cases of renal tumour.

Patients suffering from enlarged spleen may complain of a sense of weight or dragging at the site of the organ. The occurrence of perisplenitis, such as occurs over infarctions, and indeed in most diseases of the organ, may be denoted by definite pain.

Having come to the determination that the tumour in the left side of the abdomen is an enlarged spleen, the cause of its enlargement has next to be ascertained. This can only be arrived at by having recourse to the history of the case, and the concomitant clinical conditions. Examination of the blood will decide whether it is due to leukæmia or not. Associated enlargement of lymphatic glands in various regions may point to Hodgkin's disease. A malarial or syphilitic history will raise the presumption of the dependence of the splenic enlargement on these toxæmic states. The co-existence of enlargement of the liver and albuminuria, with a history of chronic suppuration, phthisis, or syphilis, will point to lardaceous disease. Cardiac dilatation, mitral disease, albuminuria, and other evidences of mechanical obstruction to the circulation—to chronic congestion; and ascites, apart from such evidences—to cirrhosis of the liver, with or without splenic anæmia.

Cysts and new growths produce irregularities on the surface, which may be detected by palpation. Their rarity hardly requires their mention here.

Treatment of Splenic Disease

The treatment of diseases of the spleen is of necessity that of the morbid conditions of which in the majority of instances they form a part; and as regards those forms in which the splenic change is a predominant feature, reference may be made to other sections of this work (see Vol. II., articles on Leukæmia, Splenic Anæmia, and Hodgkin's Disease). Abscesses and cysts, if diagnosed, may call for surgical intervention.

DISEASES OF THE LYMPHATIC GLANDS

Morbid Anatomy and Etiology

When we consider the nature and extent of the lymphatic system we cannot be surprised at the frequency with which it is involved in morbid processes. Pervading the whole body and concerned in the important office of absorption, it may be regarded as constituting an outpost of the circulation, furnishing supplies for the reinforcement of the blood, and serving also as a means of defence against its invasion by foreign elements. No small share of these duties is taken by the aggregated masses of lymphoid tissue termed the "lymphatic glands," and if for convenience we detach them in our nosology from the rest of this great system, it is only because in them morbid changes are more obvious and striking as well as more frequent than in the hardly less integral constituents of the lymphatic apparatus. Moreover, adequately to describe the pathology of the whole absorbent system would go beyond the limits of a text-book, even if it were necessary to do so for the sake of completeness. To consider separately the lesions of lymphoid structures from those of other tissues and organs would involve much repetition and perhaps not a little confusion.

The diseases of the lymphatic glands proper may, however, be usefully considered apart from those of other lymphoid organs, although even in regard to them one cannot ignore how intimately they are linked with affections of other parts. The rule is for lymphatic glands to be affected secondarily to lesions of structures

with which they are either directly in contact or with which they are connected by lymph channels. If there be any exceptions to this rule they are to be found in examples of primary malignant disease (lymphosarcoma), and perhaps in the progressive hyperplasia that constitutes Hodgkin's disease (*q.v.*), which latter, however, is now regarded by competent authorities as not entitled to rank as a primary disease of the lymphatic system.

Inflammation (Septic and Infective). — Apart from direct injury, perhaps the most common cause of acute lymphadenitis is by infective inflammation spreading through the lymphatic vessels from the primary seat of injury. There are abundant examples of this in both external and internal glands. The intensity of the glandular inflammation thus set up depends partly upon the nature of the infective agent or virus that is inoculated at the primary site, and partly also upon the individual capacity to resist or overcome the specific action of the former. Thus the effect of a poisoned wound on the finger of one individual may, *cæteris paribus*, be limited to transitory swelling of the axillary glands; in another it may produce an extensive abscess. This proneness or vulnerability to lymphatic inflammation and suppuration is especially seen in those who have inherited what is termed the "strumous" constitution, which is only another way of expressing the lack of resistance to pyogenic organisms that certain individuals possess. Whatever may be the true explanation of these variations in the degree of resistance opposed to such agencies there is nothing strange in their occurrence. A like proclivity to infection, and a like capability to resist it, connoted by the term "immunity," are recognised facts in the history of all infective disorders.

Lymphadenitis, then, may be caused by absorption of septic matter, or pyogenic organisms introduced into the subcutaneous or submucous lymphatic channels, and the full brunt of their attack is borne by those glands which are in the most direct relation to the site of inoculation. In the case of wounds of the skin the axillary glands may inflame, following any such infection from the fingers to the shoulder; a prick from a rusty needle on the finger is just as likely to be followed by axillary lymphadenitis as is the inoculation of vaccine on the upper arm. An abrasion on the foot may lead to inflammation of the inguinal glands, and so may a chancre on the penis or an urethritis. The submaxillary and cervical glands may be inflamed in scarlet fever and rubella, secondarily to the affection of the faucial mucous membrane and tonsils of those diseases; the occipital and posterior cervical groups—in impetigo or pediculosis

of the scalp. As regards internal glands, we see acute inflammation of the bronchial and mesenteric groups associated with acute bronchitis and enteritis respectively; whilst the deeper-seated retroperitoneal lumbar glands may become inflamed in consequence of inflammation of the organs (*e.g.* the testicle) whence lymph flows into them. It follows that in every case of acute lymphadenitis its cause should be diligently sought within the territory from which the inflamed glands derive their lymph.

Mention has been made of the cervical lymphadenitis of scarlet fever. This may be truly specific in some cases, but in most it is probably excited by the pyogenic organisms that, together with the scarlatinal virus, play a part in the inflammation of the throat and ear. Similarly in typhoid fever, the organism of which has a special affinity for the intestinal lymphatic apparatus, we invariably meet with more or less inflammation of mesenteric glands sequential to the necrotic ulceration of the Peyerian and solitary follicles. In diphtheria also the submaxillary and cervical groups are liable to become inflamed; and in not a few infective disorders a similar localised lymphadenitis is associated with local lesions of mucous membrane. There are, however, at least two general infections in which the glandular implication is more widespread. The one is "bubonic plague" (Vol. I. p. 128); the other, the far milder affection known as "glandular fever," first described in 1889 by E. Pfeiffer. The latter is an acute specific fever occurring in epidemics, mostly among children; and it has points of resemblance to those mild cases of generalised lymphadenitis which have been shown to be the precursor of plague, and have been termed "pestis minor." Its clinical features will be described hereafter. Suffice it to say here that so far its etiology is veiled in obscurity.

The anatomical changes of acute lymphadenitis consist in the first place of increased vascularity with exudation of lymph or fibrinous material into the lymph sinuses, and proliferation of the lymph corpuscles. Such changes explain the swelling of the gland and its macroscopic appearance, which, on section, varies from a deep reddish tint to a grayish white, according as the vascularity or the cell-proliferation predominates. The consistency of the gland is diminished. Nor is the inflammation confined to the gland substance; the capsule and the surrounding connective tissue may share in it. Besides cell-proliferation there is also cell-necrosis, which may take place in scattered foci. This change has been especially observed in diphtheria, and is closely paralleled by what takes place in the follicles of the spleen in the same disease (Oertel).

But it is met with in many other specific infections, *e.g.* in the mesenteric glands in typhoid fever, and it is associated with the liquefactive and suppurative changes that ensue on the invasion of glands by pyogenic organisms. This suppuration also may involve the periadenal tissues.

Slighter degrees of inflammation may entirely resolve, and the gland return to its normal size; or, when there has been much necrosis, the disintegrated cell elements, not being removed, may caseate and ultimately cretify. In such cases, as well as in those where resolution has been imperfect, the trabecular tissue may become increased, and the whole gland permanently enlarged and indurated.

The condition of chronic indurative inflammation may also be excited by the presence of foreign solid matter, such as dust, or tattoo-pigment, in the gland. Excellent examples of this change are to be found in the anthracosis so common in the bronchial glands of town-dwellers, and to a more marked degree in miners and stone-workers.

Tuberculosis.—The recognition of the fact that tuberculous infection of lymphatic glands is the essential feature of the conditions formerly described under the head of "scrofula" or "struma" has greatly simplified pathological doctrine, and may eventually lead to the abandonment of those nosological terms. Forty years ago, in his classical lectures on "Rickets," the late Sir W. Jenner described in vivid outline the clinical characteristics of the diatheses, "Tuberculosis" and "Scrofulosis." Nor can any one dispute the accuracy of his delineation of these clearly contrasted "constitutional" states, the features of which are just as recognisable now as they were then. Yet even this incomparable clinical observer, when he comes to discuss the subject of tuberculosis in children,[1] includes therein a description of tubercle of the lymphatic glands, which can hardly be distinguished from conditions then generally regarded as "strumous." So that, after all, the underlying identity of "tubercle" and "scrofula" must have been tacitly recognised, although not formally stated, long before Koch's discovery enabled the crucial test of this identity to be applied. The present day pathologist no longer sets store upon "diathesis," except in so far as it signifies special vulnerability. The strumous subject, with his tendency to catarrhal affections, cutaneous and glandular inflammations, is perhaps more prone than others to the inroads of bacillary infection when exposed to these;

[1] *Clinical Lectures and Essays*, 1895, p. 86, *et seq.*

and the route taken by the bacilli to the glands is probably in all cases through a damaged mucous membrane. This may be the reason why the glands which are most commonly the seat of such change are the cervical, the bronchial, and the mesenteric. The tonsils and the pharyngeal mucosa are very liable to inflammation, which, even without proceeding to ulceration, may so impair the integrity of the membranes as to render them permeable to micro-organisms; and a like liability to catarrh of the respiratory and intestinal tracts may explain the proclivity of the "scrofulous" to tuberculous infection of the thoracic and abdominal lymphatics. But apart from any such predisposition, these glands may be infected by tubercle secondarily to the lungs, or, in the case of the mesenteric glands, through ingested food or swallowed tuberculous sputa.

The main difference between the old conception of "scrofula" as accounting for these changes in lymphatic glands and the present interpretation of them is briefly this. Formerly these glandular affections were regarded as primary manifestations of a peculiar diathesis. Now they are considered as the result of specific infection in subjects specially prone to lymphatic inflammation. It is hardly possible to banish the idea of such special vulnerability, which may be inherited, and for which we may even, if it were necessary, retain the term "scrofula" or "struma"; but it is also clear that, with the knowledge we now possess of the nature of tuberculosis, it is equally impossible to look upon a "strumous gland" as the result solely of a constitutional weakness. There is in every case another and more important factor, namely, that of bacillary infection, and this must take place through recognised routes. Nor does it need much use of the scientific imagination to conceive that microbes can be taken up from apparently normal mucous surfaces into the lymphatics, since there is ample proof of similar impregnation of lymphatic glands by inorganic particles of dust—in dimensions far exceeding those of bacterial organisms—which leave no appreciable trace of their passage on the mucous membrane itself (see also Vol. I. p. 223).

It is to be noted that, although "scrofula" due to tubercle bacilli runs a milder course than clinical tuberculosis, experiments have shown that this is not due to any diminution in the virulence of the actual bacilli concerned. The difference is in the resisting power of the patient.

The *cervical glands* are the most common seats of tuberculous adenitis. The disease begins on one side, and the glands are

gradually involved in the change, which progresses slowly, with, it may be, exacerbations of a more acute character. The implicated gland becomes swollen and at first may be harder than natural, but as time goes on it may soften down, and, becoming adherent to the skin, lead to ulceration of the latter, leaving a sinus which discharges thin caseo-pus. The histology of a tuberculous gland varies with the stage of the process. In the earliest period of the infection, when the gland is but slightly enlarged, a section shows extremely well the characteristic giant-cell systems of miliary tubercle, and bacilli are to be found within it. At a later period, when the gland is larger, it presents to the naked eye opaque, firm, yellow areas which are formed by caseating cell masses. Later still the whole gland, still further enlarged, may present a uniform opaque yellow aspect, with perhaps points of softening. Here the necrotic change is still more advanced, and but little of the normal gland tissue remains. Sometimes the mass breaks down into pultaceous material. In other cases there is suppuration added to the tuberculous change, suggestive of a mixed infection with pyogenic as well as tubercular organisms, comparable to that which takes place in the lung in phthisis. In such cases the inflammatory change extends beyond the confines of the gland, and hence the adhesion of such glands to one another and to the skin, and the formation of an abscess. Or the caseous gland may remain quiescent and may become calcified. The various stages of change may often be observed in the individual glands of a chain.

The changes in the *bronchial glands* are essentially the same as those observed in the cervical, but the issues of their implication are naturally different. In the case of the bronchial glands enlargement may lead to compression of the adjacent air passages and œsophagus, and be followed by grave results. Several cases are on record in which a softened caseous gland has ulcerated into a main bronchus or the trachea, more rarely into the œsophagus, whilst compression of the vena azygos from the same cause has also been described with resulting hydrothorax. Sometimes, owing to such bronchial ulceration, secondary inhalation-tuberculosis has been set up in the portion of lung connected with the bronchus, and a not infrequent issue of such an accident is asphyxia from the escape into the trachea of a caseous plug.

The *mesenteric glands* may become tuberculous by infection from tubercular ulceration of the intestines; but, especially in children, there may be very extensive caseation of these glands, forming large conglomerate masses, apart from any such gross lesion of the

mucous membrane. Such are the cases to which the clinical term "tabes mesenterica" is applied, and their relative frequency in early life is regarded as being possibly due to the greater liability at this period to infection by ingestion of milk from tuberculous cows. Without entering into this question, which is still *sub judice*, it must suffice to point out that the only conceivable route for the infection of the mesenteric glands must be through the intestinal mucosa, and that such infection must therefore often take place without leaving any appreciable lesion in the membrane itself.

Lastly, in rare cases, tuberculosis may be widely spread throughout the lymphatic system, so much so as to simulate the progressive enlargement and general involvement of glands met with in Hodgkin's disease. In such cases it may not be possible to trace the infection to its starting-point. It is interesting to note that the spleen is here liable to be equally affected. (See also "Tuberculosis," in Vol. I. p. 220, *et seq.*).

Syphilis.—Enlargement with induration of lymphatic glands is the invariable sequel to the initial lesion of syphilis—the chancre—proving that the absorbed virus acts as an irritant on the glands in a manner analogous to that of other inoculated poisons, or the products of non-specific inflammation. It is, therefore, the glands which are in connection through lymphatics with the primary sore that exhibit the effects as well as adduce the proof of syphilitic inoculation. Having regard to the usual site of the primary lesion, the inguinal glands are those which are most frequently so affected, the glands enlarging within a few days of the inoculation in a slow and progressive manner, until the whole group may be involved. The affected glands remain discrete, and are generally smooth and hard. They may continue enlarged for months and then slowly subside. Examination of such glands does not reveal anything of a specific or pathognomonic character. They are the seat of increased cell-proliferation—a lymphadenitis, in fact—which never goes on to suppuration. It is rather in their clinical than in their pathological characters that they are distinctive, although even here it must not be forgotten that inguinal glands may become enlarged in a similar manner by other forms of irritation, *e.g.* as the result of excess in walking. The concatenate glands are also apt to remain enlarged for many months in syphilis.

Besides the lymphadenitis of primary syphilis, there is also that which arises from local irritation in connection with the ulcerative lesions of the later periods of the disease, and, moreover, the

characteristic gummata of these stages have been known to occur in lymphatic glands as in other organs.

Lardaceous degeneration does not spare lymphatic glands, rendering them swollen, firm, and glistening on section. The change is most pronounced in the fine reticulum of the gland, which becomes irregularly swollen and is eventually converted into masses of lardaceous substance. At the same time the lymphoid corpuscles diminish in number. In most cases of advanced lardaceous disease where the viscera are largely implicated, the lymphatic glands will be found to be involved in the change (Vol. II. p. 101).

Malignant disease occurs in the two main divisions of sarcoma and carcinoma, but the latter only as a secondary or metastatic growth. Sarcoma may be either primary or secondary. The most usual form of primary tumour is a small round-celled growth in which a delicate stroma may be traced, and it is therefore often termed "lymphosarcoma." It is most often met with in the intrathoracic glands, forming the chief kind of solid mediastinal tumour. Spindle-celled growths and the variety termed "alveolar sarcoma" may also arise primarily in glands; but melanotic sarcoma is invariably secondary. It may be mentioned that not all the secondary glandular growths of melanotic sarcoma are pigmented, and that if this variety be excluded it will be found that sarcomata in general are not prone to metastatic dissemination in lymphatic glands. In this respect they differ from the *carcinomata* which seem especially liable to invade the lymphatic system. Glands which are in relation by their lymphatics to cancerous growths are apparently very early infected. They may be almost unchanged to the naked eye, and yet found microscopically to contain the early epithelioid elements within the lymph sinuses, the germs of which must have been conveyed from the primary growth. When the gland is fully infiltrated it reproduces all the characters of the original formation, so that an axillary gland in a case of mammary scirrhus becomes dense, hard, and converted into a typical mass of carcinoma; whilst a submaxillary gland in one of epithelioma of the tongue or lip will be whitish and friable, and yield on scraping abundant cell-nests characteristic of such a growth.

Clinical Characters

The clinical features of lymphatic gland disease have been for the most part described elsewhere, for, like the spleen, the lesions of

lymphatic glands mostly form only one of the group of symptoms of a general disease. Such affections as Hodgkin's disease and mediastinal tumours have also been separately considered. It only remains to refer to the clinical characters of so-called "glandular fever," and of lymphatic tuberculosis or scrofula.

GLANDULAR FEVER.—The comparatively scanty literature upon the subject, whilst doubtless denoting the rarity of its occurrence, leaves no doubt as to its reality and distinctive nosological position. It is a highly infectious malady, characterised by inflammatory swelling of lymphatic glands, running an acutely febrile course, and followed by somewhat slow convalescence marked by anæmia. It resembles other acute specific fevers in its course, which is fairly definite. Its subjects are almost invariably children, and its contagiousness is quite as marked as that of measles or mumps, so that if one child in a family be attacked the others rarely escape.

The incubation period is from five to fifteen, usually six or seven days, and the distinctive feature of the disease, lymphadenitis, which takes the place of an exanthem, commences within two days of the onset of febrile symptoms. This onset is sudden, marked by headache, nausea, or vomiting, a rise of temperature ranging from 101° to 105°, a rapid pulse, and accelerated respiration rate. Together with this there are pain and tenderness on one side of the neck over the anterior triangle, the head being turned to the painful side. The tongue is furred and moist, the bowels generally constipated. There may also be some abdominal pain. There is no complaint of sore throat and no rash on the skin. It is generally said that no change is to be observed in the throat, but one writer (L. Durno), in describing an epidemic, observed "in nearly every case a deep purplish blush over the mucous membrane of the fauces, tonsil, and pharynx on the affected side."

At a period varying from twelve to thirty-six hours evident swelling of cervical lymphatic glands appears, the glands being those seated beneath and in front of the sterno-mastoid, and generally on the left side, it taking about two to three days for the whole chain to be implicated. The swelling is confined to the individual glands, and there is no redness of the skin, nor does the inflammation ever pass on to suppuration. In from two to four days the inflammation begins to subside, and in the milder cases no other glands are involved. In the majority, however, the corresponding glands on the other side of the neck undergo a similar progressive inflammation, and in a certain proportion of these the change involves several other gland-groups in various parts of the body, *e.g.* the axillary and

inguinal, and also, no doubt, the mesenteric, accounting for the abdominal tenderness. In such cases it may be three weeks before all evidence of this lymphadenitis has subsided. Consentaneously with the lymphatic affection there is, in most cases, evidence of enlargement of the liver and spleen, with tenderness over these organs. Constipation is the rule, but this may be followed by mucous stools, sometimes in marked degree, diarrhœa occurring at the approach of convalescence. In a few cases hæmaturia pointing to nephritis has occurred as a complication. The pyrexia, which may be high, persists so long as the signs of glandular inflammation last.

Anæmia and depression mark the convalescence, which takes place gradually. Death is a termination of the greatest rarity.

There can be no doubt that we have here to do with an acute specific disorder, which somewhat resembles "pestis minor," but so far its cause remains unknown. Durno thinks the throat symptoms he observed suffice to point to the seat of entrance of the infective agent, whilst others who have not noticed any faucial change are inclined to regard the intestinal tract as the site. Certainly, whatever its origin, it is highly infectious, and invariably occurs in limited epidemics.

The **treatment** employed is mainly symptomatic. Salicylate of soda was give by Durno, and small doses of calomel for the constipation. Rest in bed with liquid nourishment is essential during the febrile period. The convalescence must be aided by change of air and the administration of such tonics as cod-liver oil, iron, and arsenic.

Scrofula is the term applied from time immemorial to a condition in which chronic enlargement and caseation of lymphatic glands are the essential features. We have seen that the glandular change in its most typical form is tuberculous in nature, and that, therefore, there is no such thing as "scrofula" regarded pathologically. Clinically it is different, since by the term scrofula is understood not merely the final stage of the glandular changes, but the antecedent conditions also. It is still open to believe that in the "scrofulous," inflammatory affections of skin and mucous membranes conform to a special type with greater liability to secondary glandular inflammation than in other individuals; but the essential feature of such cases is the readiness with which they are liable to be infected by tubercle. In other words, the tendency of scrofula is towards tuberculisation.

The clinical expression "scrofula" may thus be taken to con-

note a condition met with in childhood in which the lesions, though not in themselves specific, are characterised by their multiplicity, their intractability, and their liability to recur. The typical affections are inflammation of the skin, mucous membranes, and eyes, eczema, catarrh of the middle ear, nose, and deeper respiratory organs, vaginal discharges (gonorrhœa and tubercle being excluded), and swelling of glands. Cornet in his admirable monograph distinguishes two forms, the tuberculous and the non-tuberculous, the latter being caused by the toxins of pyogenic cocci. The latter is, as has already been said, extremely liable to pass into the former owing to secondary infection with tubercle bacilli. The glandular swelling in the pyogenic variety is usually transitory (*cf.* the enlargement of the inguinal glands in gonorrhœa), but the cocci sometimes settle in the glands and lead to the formation of abscesses. On the whole the prognosis of this form is favourable unless tubercle supervene. The tuberculous variety passes through four stages. In the first the affection is superficial, involving the skin and mucous membranes; in the second the glands are implicated; in the third the bones and joints suffer as well, while the last phase is general and often complicated by amyloid disease.

Scrofulous glands in general are characterised by the steady formation of large masses in which the outlines of the individual glands become indistinguishable. Their resistance varies; they are at first hard, later soft, and then fluctuate. Finally they become adherent to the skin.

The clinical history of the most common form of such glandular infection is that in which the *cervical* glands are involved, especially those of the submaxillary region and in the anterior triangle. The enlargement generally begins insidiously and painlessly, and is not necessarily preceded by any obviously inflammatory condition of skin or mucous membrane. On the other hand the child—for the disease is in great measure one of childhood—may have been the subject of chronic naso-pharyngeal catarrh, or otorrhœa, or impetigo of the scalp, or, often, of enlarged tonsils. At first and not seldom throughout, the glandular change is unilateral; but if both sides are involved it is more on one side than another. The enlarged glands are firm, smooth, and discrete, but in course of time the glands of the chain which are the most affected become adherent to one another, forming an irregular mass. Then adhesion to the overlying skin may take place, and evidence of the breaking down of a gland be afforded by its becoming soft and fluctuating. For, unlike the change in Hodgkin's disease, there is a liability to

suppuration, which has been explained by assuming that the infection is "mixed," the tubercle bacilli being associated with pyogenic microbes. When the disease has reached this stage—and it may take months or even years to do so—the skin over the most advanced glandular abscess breaks down, and the caseo-purulent contents of the latter are discharged, leaving ragged sinuses which take long to heal, with resulting unsightly deeply puckered scars. The modern practice of having recourse to surgical intervention before the glands have suppurated is rendering such disfigurement less common than formerly, a linear cicatrix alone remaining to mark the site of the incision through which the chain of diseased glands was removed. Some of the glands may have become cretified, and therefore obsolescent, but the *rationale* for their removal is based as much on the ground that they constitute a focus for general systemic infection, as it is to avoid unsightly scarring. There is great variation in the rate at which the process takes place, and when it is very active it is accompanied by fever. When the disease extends to other groups of glands, such as the posterior cervical and axillary, or those of the opposite side of the neck, the case may be most difficult to distinguish clinically from one of Hodgkin's disease.

Enlargement of the *bronchial* glands, although one of the most common pathological lesions, often remains undetected during life. In those cases—again most common in children—where they become the seat of tubercle and pass through caseation and even suppuration without any apparent permanent change in lungs or air passages to account for them, the presence of the change may be unsuspected until quite late, when signs of pressure upon the important structures in their proximity give rise to the inference. Thus attacks of paroxysmal coughing and of dyspnœa (sometimes simulating asthmatic seizures) may be due to pressure upon the trachea or the vagus nerve. These signs may precede a fatal attack of asphyxia due to ulceration of a suppurating gland into the air passage. Perforations of the œsophagus or of the aorta have also been known to occur, but very rarely indeed. Direct evidence of this gland affection may be obtained by physical examination, in percussion dulness over the manubrium sterni in front and inter-scapular region behind, with intensified tubular breathing over the dull area. It must also be borne in mind that bronchial gland tuberculosis may occur as a remote sequel of measles and whooping-cough.

The subject of *mesenteric* tubercle (tabes mesenterica), which is

especially frequent in infants, is pallid and emaciated. The large abdomen contrasts with the puny size of the rest of the body. Irregularity of bowels with loose and fœtid stools is common. It is sometimes possible to feel the enlarged glands through the abdominal wall, but the flatulent distension of the intestines may be a bar to this. Nor is the disease confined to the mesenteric glands; the great chain behind the peritoneum may also be implicated, forming a firm and fixed resisting mass.

Prophylaxis.—In the contest against scrofula prophylaxis must, of course, play the leading part. Its main agents may be summed up in the three words *light*, *air*, and *cleanliness*. These are habitually regarded by the poor as the chief enemies of childhood, and the campaign against this form of prejudice makes but slow headway in our over-crowded cities. Especial attention should be paid to keeping the head clean and to washing round the mouth, particularly after the consumption of saccharine substances. Small excoriations should not be neglected, but should receive prompt attention. As to nourishment, infants cannot, of course, be better than breast-fed by a healthy mother. Failing this scalded standardised cow's milk is to be recommended. In suspicious cases the date of weaning may with advantage be deferred. The child should be early rendered familiar with the external use of cold water; the cold douche or bath is a "hardening" agent of the greatest value. Particular care should be exercised in protecting weakly children from household epidemics. Many a lingering illness dates from the delayed termination of an attack of measles or whooping-cough. Finally, every care must be taken to avoid overcrowding and over-pressure at school.

Apart from extirpation, which obviously has its limits, the **treatment** of these glandular affections does not differ in its general principles from that adopted for other forms of tuberculosis. Fresh air is an essential element, and in this regard the bracing climate of the Isle of Thanet has long had a well-deserved reputation. Wholesome food is another adjuvant, and in this category may be included cod-liver oil. The aim is to improve the general nutrition, and thus promote the obsolescence of the tubercle. That such comparative "cures" of these local tuberculoses do take place is evidenced by the calcification of internal glands that may be found in the bodies of adults dying from other diseases. This line of treatment is also prophylactic; it may do much to lessen the vulnerability of the "scrofulous" to bacillary invasion, possibly by stimulating phagocytes to get rid of the tuberculous agents.

CHLOROMA

This disease was first described by Dr. Allan Burns in 1823, since which date not more than about thirty cases have been recognised. There seems to be no doubt that the affection is a definite clinical entity, but its rarity renders the precise description somewhat difficult. It is probable that not all the cases enumerated under this heading can be accepted as true chloroma.

Etiology.—Chloroma appears to be a disease occurring in childhood, youth, and early middle life, and attacking by preference the male sex. It is characterised by green tumours in different parts of the body, and by a blood change, similar to that seen in lymphatic leukæmia.

Morbid anatomy.—It is now generally supposed that the tumours are of a lymphosarcomatous nature, and take their origin primarily in the periosteum, usually of the skull bones; it is possible, however, that further research may result in showing that they are the lymphoid tumours ordinarily seen in leukæmia which have acquired a green colour as an epiphenomenon. The green colour at first deepens on exposure to the air and then fades, but can be restored by the addition of peroxide of hydrogen; it is most probably due to a fatty pigment (lipochrome). The chief sites of the tumours are in the orbits, skull bones, vertebral column, gums, palate, spleen, kidneys, and lymphatic glands and bone marrow. In some cases it appears that not all the tumours are pigmented. The bronchial and mesenteric glands are almost always enlarged, the cervical glands less often. In one case, at least, in which the kidneys were involved the urine had a greenish tint.

The blood change is always of the nature of a lymphæmia. There may not at first be an actual excess of white blood corpuscles, but there is always a relative increase of the lymphocytes. It appears that green tumours may be seen when the typical disease cannot be demonstrated. Thus in a case recently recorded by Türk, the patient died of splenomedullary leukæmia, and it was found at the post-mortem that the characteristic lymphoid deposits had a green colour. Bramwell has gone so far as to suggest that cases of true chloroma may occur without the green pigmentation.

Clinical course.—The patient, as a rule, appears at first to be suffering merely from anæmia, or there may be only a general malaise. In a typical case the next symptom is exophthalmos, which may be unilateral or bilateral, and is due to the development

of the tumour in the orbit. Swellings in the temporal region now often appear, and are caused by growths in the temporal fossæ. In this way an unmistakable facies is developed. The hearing is often affected; tinnitus and deafness have been recorded in at least one-third of the cases. Headache and giddiness often occur, and the patient passes into a drowsy, apathetic condition which continues until death. This has been the inevitable result in all cases hitherto recorded; it usually occurs within six months, but patients have been known to live as long as two years. The diagnosis in a typical case is not difficult, the characteristic tumour and the lymphæmia being sufficient.

When the exophthalmos is the first sign the disease has to be distinguished from exophthalmic goitre, and from the various kinds of orbital tumours; examination of the blood in such a case should clear up the difficulty.

No satisfactory **treatment** has as yet been devised.

DISEASES OF THE THYMUS GLAND

The thymus gland attains its maximum development about the second year of life, after which it gradually shrinks, until at puberty little remains but a small mass of fatty tissue, within which may be found scattered areas of lymphoid corpuscles and a few relics of the primary epithelial elements in the form of Hassall's "concentric corpuscles." Occasionally, however, the gland persists beyond this period, and many cases of mediastinal tumour have been attributed to new growth arising within it.

The most interesting and important subject connected with the thymus gland is that of certain cases of paroxysmal dyspnœa known as "thymic asthma," in which the gland is found to be manifestly hypertrophied. The attacks, by some regarded as of the nature of "laryngismus stridulus," are attributed to the pressure exerted on the trachea by the enlarged organ, and a similar explanation has been assigned to the occurrence of sudden death in infants in whom the thymus has been found to be of large size. These cases of sudden death fall under two categories. In the one the enlarged thymus is a part of the status lymphaticus (see p. 645); here it would appear that the enlargement is not, strictly speaking, the cause of death, but is rather one of the indications of a condition in which the enfeebled resisting power may lead to a lethal issue of apparently trivial incidents. In the other the hypertrophied gland

may press on the heart or vagi and so occasion cardiac failure. There is yet another class in which it may cause mechanical compression of the trachea; here death occurs from asphyxia, the heart continuing to beat after respiration has ceased. In this instance death is not, strictly speaking, sudden, and does not occur without warning.

In connection with the first group it is instructive to note that Dr. Osler refers to cases described by various writers of sudden deaths in adults "occurring under such conditions as anæsthesia or while bathing. In a number of these cases not only has the thymus been found enlarged, but the spleen and lymphatic tissues generally."

Persistence and enlargement of the thymus has been observed in cases of exophthalmic goitre, leukæmia, myxœdema, Addison's disease, and acromegaly. No satisfactory explanation of this coincidence has been afforded, although in the case of Graves' disease the association is apparently fairly constant. Discordant opinions have been expressed as to the use of thymus extract in this disease, both as to its efficacy and the *rationale* of its prescription. On the latter point some think the enlargement is compensatory to the thyroid change, but others, with equal reason, fail to see how its administration in this affection can be efficacious, since the gland is itself enlarged in it.

Of morbid conditions, other than hypertrophy, there are few of any consequence, except malignant growths, both sarcomata and carcinomata. These probably account for most of the new growths that arise in the anterior mediastinum. Their presence is denoted by well-marked physical signs, and by evidence of compression of the superior cava and innominate veins. Mediastinal tumours of this class have to be distinguished from aneurysms of the aortic arch, which they may simulate. The growth may extend downwards to the pericardium and give rise to hæmorrhagic pericarditis.

For the rest, inflammation and suppuration are rare events. Tubercle has been met with, and softening gummata or abscesses have been described in congenital syphilis.

DISEASES OF THE THYROID GLAND

Morbid Anatomy and Etiology

Acute thyroiditis.—This is a rare condition, which has been met with in various infective and septic diseases, such as enteric

fever, diphtheria, influenza, malaria, puerperal fever, variola, and pyæmia. It has also been described as a complication of acute rheumatism. The age incidence is most commonly between twenty and forty years, and the female sex is the more frequently affected.

The affection is usually streptococcal, but the pneumonia and typhoid bacilli have also been found in connection with it. Sixty to seventy per cent of the cases suppurate, about one-fourth clear up, a few become gangrenous.

The mortality is about 25 per cent; the chief danger is rupture of the abscess into a vital part.

Goitre.—A complete survey of the diseases of the thyroid would embrace the conditions of cretinism, myxœdema, and exophthalmic goitre, which have been already described (see Vol. II.). Pathologically speaking these affections are of greater importance than the much more common conditions to which the generic term of "goitre" is applied, and for the following reason: cretinism represents the effects produced on the organism by the removal or atrophy of the gland, whilst exophthalmic goitre is characterised by symptoms that may be regarded as due to an excessive production of the normal thyroid secretion. In ordinary goitre, on the other hand, whether this be endemic or sporadic, no such characteristic symptoms of functional derangement are necessarily exhibited. The symptoms are mainly those which can be ascribed to the mechanical pressure of the enlarged organ.

It is obvious that enlargement of the thyroid may take place in many ways, and although custom has sanctioned the application of the term "goitre" to them all, it is, to say the least, unlikely that such varied pathological conditions can be initiated by the same influences. Broadly speaking, those anatomical varieties which arise endemically in so-called "goitrous districts," such as the "Derbyshire wen," are of the nature of follicular hypertrophy, with cystic or fibrous degeneration, whilst many of the sporadic cases include new growths, such as adenomata and carcinomata.

(*a*) *Acute.*—This curious affection appears to occur chiefly in the form of epidemics among French soldiers. A very good account has been published by Dr. Ferrier of Val de Grâce.

The right lobe is usually the more affected. The principal associated symptoms are bradycardia, loss of weight, and at first some hypothermia. The general condition is good, and there are very few nervous symptoms. The goitre usually disappears under treatment, but may undergo cystic degeneration. There is thus a general resemblance to endemic goitre. In both the functional

activity of the gland is diminished, and both probably arise from some generally infective cause. No micro-organisms have been found in either. The most satisfactory remedy for acute goitre appears to be thyroid extract, under the influence of which the gland diminishes in size and the pulse accelerates. Iodine is of use in some cases, but thymus extract appears to be valueless.

(*b*) *Chronic.*—Much has been written concerning the etiology of endemic goitre, and various theories have been propounded to explain its prevalence within the comparatively limited areas in which it is found. Such districts, both in this country and abroad, are often mountainous, the disease occurring amongst the dwellers in the valleys rather than those of the highlands. Nor is goitre confined to those who are born in the district, although with them whole families may be the subjects of it, the goitre developing at almost any age from childhood to middle life. But new settlers are liable to become affected, and even the domestic animals. To account for such a distribution, and also for the apparent capriciousness with which goitrous districts are scattered, the course must be looked for either in the soil or the drinking water. Geologically speaking, endemic goitre is only found to occur upon marine deposits, especially palæozoic, triassic, and tertiary. All fresh water and eruptive formations, as well as certain sea-water deposits (*e.g.* Jurassic), are free. Thus in England goitre is only endemic upon chalk and sandstone. The view that the goitre results from the consumption of waters laden with salts of lime and magnesia is now generally abandoned. At the same time it is almost universally admitted that the drinking water is in fault, not, however, owing to its chemical composition, but to its acting as the medium by which the contagium is transmitted. This view was long ago advanced by Virchow, who suggested a miasmatic origin, comparable to that of malaria. Ewald holds that there is probably a pathogenic micro-organism which requires certain conditions in the drinking water for its extra-corporeal life, and a particular predisposition also in its human host. Such microbes have been described by Italian observers, but the discovery lacks confirmation.

There is a manifest relationship between cretinism and goitre, the two conditions prevailing together in districts where goitre is endemic. Many cretins are goitrous, and the children of goitrous persons are often cretins; yet it has been shown that this latter condition does not arise if the goitrous have removed from the endemic area. It would, therefore, seem as if the same poisonous influence which produces goitre in the parent is liable to affect the

offspring congenitally. When acquired, endemic goitre usually appears at about the age of puberty. The female sex is the more often affected.

In sporadic goitre divers etiological factors, such as age, occupation (involving stooping), pregnancy, injury, and other causes of hyperæmia of the gland, must be taken into consideration.

The most common form of goitre is the *parenchymatous*, where the gland is enlarged in all its parts. This enlargement may involve both lobes symmetrically, or one side may be much more enlarged than the other. There is a general hypertrophy of its substance, the vesicles are more filled with their colloid contents, and the stroma is also increased. In some cases the contents of the vesicles become more fluid, and several vesicles may coalesce to form cysts. This *cystic* goitre may be carried to an extreme degree, and involve not only the vesicular structures, but the more solid parts of the gland. A *fibrous* goitre is one in which there is an excessive development of the interstitial tissue of the organ.

New growths.—*Adenoma*, or "adenomatous goitre," consists in the development within an enlarged thyroid of variously sized encapsuled tumours, which in structure conform more or less to the normal type of thyroid gland tissue. Berry distinguishes two sub-varieties, namely, (*a*) fœtal, and (*b*) cystic adenoma. The fœtal adenoma occurs chiefly in children and young adults, and is composed of tissue resembling that found in the fœtal thyroid. It is markedly vascular, and has a solid, homogeneous appearance, being composed chiefly of epithelial cells resembling undeveloped thyroid vesicles. The cystic adenoma is much more common, and is beset with numerous cavities formed by the coalescence of vesicles with colloid and often hæmorrhagic contents. There is also much connective tissue.

Carcinoma and *sarcoma* are also liable to occur in the thyroid. It is said that malignant disease is more prone to develop in a gland that has already been the seat of innocent goitre, and that this is why it is more common in districts where ordinary goitre is prevalent (Berry). It is also more common after pregnancy, in which the gland is always enlarged. Sometimes in cancerous metastases thyroidal tissue appears (so-called malignant adenoma).

Tuberculosis.—Miliary tubercles have been met with in the thyroid in cases of acute general tuberculosis. They are mostly in the form of gray granulations. Caseation is very unusual.

Syphilis.—The thyroid is said frequently to undergo slight general enlargement in this disease; but gummata are rare (Berry).

Cysts.—In addition to simple cysts, which occur in multiple form in well-marked cases of goitre, it is not uncommon, especially in elderly subjects, for cysts, sometimes with hæmorrhagic contents, to form, from the breaking down of the walls of vesicles. The walls of the cysts may become calcified. Echinococcus cysts have also been met with.

Before leaving this subject reference must be made to the "parathyroids," or "accessory thyroids," which are occasionally developed in the vicinity of the main gland. They are interesting from the fact that, like the thyroid itself, they may become enlarged and also be the seat of morbid growth. Recent research has shown that the fatal consequences of thyroidectomy in animals only follow if the parathyroids are excised as well. It appears that removal of these glands alone is even more fatal than that of the thyroids. Attempts have been made to differentiate the symptoms of myxœdema into thyroidal and parathyroidal, but hitherto without definite result.

Reference must also be made to the therapeutic importance of the recent discovery in the thyroid and parathyroid of iodine and arsenic as normal constituents. See article "Disorders of Metabolism" by one of us (B. A.) in *Medical Annual* for 1902.

Clinical History and Treatment

For full descriptions of the clinical features of thyroid affections and their treatment, reference must be made to surgical treatises. In this place only a bare outline of these subjects can be given.

Acute thyroiditis is characterised by painful swelling of the gland, accompanied by pyrexia. The swelling is hard and tender to touch at first; but, as frequently happens, it may become in places soft and fluctuating from the supervention of suppuration. There is danger to life from asphyxia from compression of the trachea, should the inflammatory swelling rapidly develop. Indications of such compression are stridor, metallic cough, and dyspnœa. In some cases there may also be dysphagia.

Treatment is mainly local: cold applications, and, if signs of suppuration occur, the opening of the abscess and drainage. In rheumatic cases the administration of salicylates is advisable. A mild purge should always be given: Dover's powder is of value in the relief of pain.

The enlarged thyroid or goitre forms a tumour in front of the

neck, which may extend almost from the angle of the jaw to the sternum. It is smooth and may be fairly symmetrical, or one lobe may be obviously much larger than the other. When situated to either side it may displace the trachea, and in any case it will probably push the great vessels and the sterno-mastoid muscle outwards. Owing to its relation to the larynx it will rise and fall with this structure in the act of swallowing, unless it be of large size or fixed to surrounding parts. Dysphagia may occur, but is not common. Serious symptoms may be caused by compression and displacement of the trachea or the laryngeal nerves, and the rare event of sudden death, which has sometimes occurred, has been attributed to pressure on the vagi. Sometimes the onset of dyspnœa is almost sudden, and may be fatal from the rapid increase in size of the bronchocele. Such fatalities are more common in young subjects (Berry), perhaps because in them the trachea is more yielding, and in the substernal variety (*goitre plongeant*) owing to its position.

Malignant disease may be suspected if a goitre which has long been present begin to enlarge in later adult life, or if a tumour first shows itself at this period. The malignant growth is also denser than the innocent and feels very hard. It may grow rapidly, causing considerable pain ; and as it extends may come to involve surrounding parts and be bound down to them. Diagnosis may be excessively difficult when the enlargement, whether malignant or not, occurs in the accessory thyroid.

In regard to **treatment** it has been found that removal from the locality where goitre is endemic is sometimes followed by cure. Since the disease is considered to be induced through drinking impure water, it has been suggested that in goitrous districts all water used for this purpose should be previously boiled. Medicinally, iodine and its salts have long had the reputation of influencing the growth of bronchocele. The tumour may be painted over with tincture of iodine or biniodide of mercury ointment, and iodide of potassium be administered internally. Thyroid extract has been prescribed in some cases in which iodine has failed, with apparently good result. Many other drugs, such as arsenic, mercury, and strychnine, as well as thymus extract, have also been given. Surgical measures, such as tapping of cysts, bronchoceles, injections of iodine into the solid tumour, and radical extirpation are also had recourse to.

A hypertrophied thyroid is even more liable than a normal one to become inflamed in the course of infectious diseases. Suppura-

tion of the gland is hence not uncommon when typhoid attacks a goitrous subject; the prognosis is very grave, the treatment is incision.

DISEASES OF THE SUPRARENAL CAPSULES

The morbid conditions of the suprarenal bodies other than those met with in cases of Addison's disease are numerous, but of comparatively slight importance clinically. Perhaps the most remarkable fact in adrenal pathology is that the "symptom-complex" known as Addison's disease is almost entirely confined to one particular kind of destructive lesion, namely, a fibro-caseous conversion of the capsules of tuberculous origin. It is true that in rare instances the clinical features of that affection have been met with where such tuberculous destruction of the capsules has not existed, in cases of atrophy, of hæmorrhage, and of malignant disease. But not only are such cases rare as examples of Addison's disease; they are rare also as denoting during life the adrenal change. For it may be safely asserted that the vast majority of examples of these morbid changes are only revealed at the post-mortem examination, not having presented during life any distinctive symptoms, and this notwithstanding the fact that the lesions may be so extensive as to have wholly destroyed both capsules.

Some of the changes met with *post-mortem* in these organs are due to their natural involution. Thus, as age advances, the adrenals generally dwindle in size, and their cortical cells become pale, granular, and fattily degenerated. They lose the firmness characteristic of the gland in youth; so that unless great care be taken they readily rupture in the attempt at their removal from the body. Owing to this fatty degeneration the cortical layers become yellowish in colour. *Atrophy* may, however, occur quite early in life or comparatively early, and must then be regarded as either due to lack of development or to a morbid process. Cases have been described, indeed, where the glands have been congenitally absent, this being usually associated with other developmental anomalies, especially in the brain. Thus anencephalous fœtuses may have no suprarenals, and it is said that the absence of these bodies is especially connected with absence of the anterior portions of the cerebral hemisphere (Zander). In the case of a man who died from Addison's disease at the age of thirty-four, the right capsule was represented by a few grains of orange-coloured substance,

and the left one was reduced to one-third the normal size (*Path. Trans.* vol. xxxvi.); and other similar instances of this rare association have been published.

Hæmorrhage into the capsules is not very uncommon. No doubt this is sometimes traumatic, as for instance when found in the bodies of still-born infants after difficult labour (Dr. H. Spencer). At other times it appears to be spontaneous,—"suprarenal apoplexy," as it has been termed,—and a remarkable instance of this was observed by one of us (S. C.). Here the right adrenal contained a recent blood clot of the size of half a walnut, whilst the left was wholly replaced by a large encapsuled hæmatoma adherent to the kidney and fully four times the size of the normal capsule. The late Dr. Greenhow, who records the case (*Path. Trans.* vol. xxviii.), was inclined to think it one of an early stage of Addison's disease, but this view is open to question. Some of these cases of suprarenal apoplexy may be due to rupture of vessels in soft and vascular new growths. Suprarenal hæmorrhage is not infrequently seen in post-mortems upon cases of acute septicæmia, and has also been found as a complication in miliary tuberculosis, bronchopneumonia, and other diseases of the respiratory organs; it is constantly seen in guinea-pigs killed by the injection of typhoid, diphtheria, and certain other bacilli. It has been described in yet another class of cases (*Path. Trans.* 1898, p. 255), wherein the patient, a child, dies after an acute illness lasting two or three days and usually associated with a purpuric or bullous eruption. In this instance the suprarenal hæmorrhage appears to be part of the actual disease, which according to some is an intestinal toxæmia.

In cases of *lardaceous disease* the suprarenal capsules are often the seat of the infiltration. They then acquire an increased consistency, and present, on section, the characteristic waxy appearance, the change in advanced cases being very marked in the cortex and in the vessels of the medullary portion.

Miliary *tubercles* may be met with in cases of acute generalised tuberculosis; whilst caseous nodules, sometimes of large size, have been found apart from any symptoms of Addison's disease during life; although, as already stated, in the majority of examples of that affection the fibro-caseous change is undoubtedly tuberculous in character. *Gumma* of the suprarenal is of occasional occurrence.

In respect to new growths, the most interesting is *adenoma*, in which there is an atypical overgrowth of masses of epithelioid cells, separated by stroma, which recall the normal cell columns of the cortex. These elements readily undergo fatty degeneration, and

the growths themselves were termed by Virchow, "*struma lipomatosa suprarenalis.*" They are now regarded as sarcomata of the vascular perithelium (peritheliomata). They are apt to become converted into hæmorrhagic cysts; if this degeneration do not occur they may tend to become clinically malignant. Primary and secondary malignant growths also occur, both *sarcoma* and *carcinoma*. Primary growths are not common, and, like renal growths, are more frequent in children than in adults. They sometimes attain a large size, and clinically simulate renal tumours, owing to their close anatomical relation to the kidney. They are friable, bleed readily, are closely adherent to surrounding parts, and frequently attack both capsules simultaneously.

Lastly, it may be mentioned that, as with the thyroid, so with the suprarenal, there may occur "accessory suprarenals," which may themselves become the starting-points of neoplasms. Moreover, new growths of the kidney may arise from suprarenal "rests" in that organ.

During the past few years much attention has been devoted to the chemical and physiological properties of extracts of the suprarenal capsules. It has been shown that preparations of the medullary portion only of the organ are physiologically active. A striking fact is the smallness of the dose required; a maximal effect results from the injection of half a milligram of suprarenal extract per kilo of body weight. That the capsules are essential to life was shown long ago by Brown-Séquard; the properties of their internal secretion have been carefully studied by many recent observers, whose results are in general, if not in detailed, agreement.

Injection of suprarenal extract produces three main phenomena: an enormous, though transient, rise of blood pressure; slowing and strengthening of the heart and pulse; and prolongation of muscular contraction. The first effect is probably due to peripheral stimulation (of the arterial muscle-fibres, Schäfer and Oliver; of ganglia, Gottlieb), while it is not yet settled whether the second results from excitation of the vagus centres or of the intracardiac ganglia. There can, however, be little doubt that the internal secretion of the suprarenals regulates the tone of involuntary muscular tissue, particularly that of the heart and blood-vessels. Cybulski holds that it exerts a specific influence upon the respiratory centre as well.

The active principle has been the subject of careful chemical studies by Sigmund Fraenkel and by Abel. The former isolated a non-crystallisable body which he called sphygmogenin, from which pyrocatechin could be obtained by boiling with hydrochloric acid.

The latter went farther, and found that the active properties resided in a benzoate having the formula $C_{17}H_{15}NO_4$; to this he has given the name of epinephrin. Takamine's adrenalin, which is now so extensively employed, is, according to Abel, simply an impure epinephrin.

Therapeutically, suprarenal extracts and preparations exercise no striking influence upon metabolism; their alleged utility in rickets has failed of confirmation. Their value depends mainly upon their vaso-constrictor power; they are, accordingly, much used for the arrest of hæmorrhage, *e.g.* epistaxis, in the treatment of hay-fever and, in association with eucaine, to render bloodless the field of small operations.

DISEASES OF THE PITUITARY BODY

Although little is known with certainty regarding the function of the pituitary body, yet its structure conforms somewhat strikingly to that of the thyroid and adrenals, its two lobes being composed of follicles containing epithelioid cells, and invested by a stroma of delicate connective tissue. Moreover, the curious fact that in most cases of acromegaly that have been examined after death, this gland is found to be hypertrophied or the seat of a new growth, goes far to suggest a comparison of the relation in which it stands to that condition, with that in which the thyroid stands to myxœdema.

Adenomatous cysts, *fibrous* and *sarcomatous* growths have been met with, and when of large size the tumour or cyst may cause atrophy of the optic nerves by compression, and also considerably compress the cerebral cortex in the vicinity of the olfactory nerves. One of the most characteristic symptoms is bitemporal hemianopsia, and mental change may be conspicuous in advanced cases. Dr. Rolleston has described a case of acute acromegaly caused by sarcoma of the pituitary body, while Dr. William Hunter, on the other hand, records a good-sized gumma of that organ which had given no symptoms during life.

Sidney Coupland.
Bertram Abrahams.

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